ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY

FOUNDED BY JAMES PLEASANT PARKER

VOLUME 54

Editor

ARTHUR W. PROETZ, M.D.,
Beaumont Building, St. Louis, 8

Associate Editor

BERNARD J. McMAHON, M.D.
Missouri Theatre Building, St. Louis, 3

Editorial Board

T. E. CARMODY, M.D. . . . . *Denver*
SAMUEL J. CROWE, M.D. . . . *Baltimore*
W. E. GROVE, M.D. . . . . *Milwaukee*
DAVID HIGBEE, M.D. . . . . *San Diego*
ANDERSON C. HILDING, M.D. . . *Duluth*
FREDERICK T. HILL, M.D. . *Waterville, Me.*
MARVIN F. JONES, M.D. . . . *New York*
HAROLD I. LILLIE, M.D. . *Rochester, Minn.*
JOHN G. McLAURIN, M.D. . . . . *Dallas*
LEROY A. SCHALL, M.D. . . . . *Boston*
H. MARSHALL TAYLOR, M.D., *Jacksonville, Fla.*
O. E. VAN ALYEA, M.D. . . . . *Chicago*

Published Quarterly

BY THE

ANNALS PUBLISHING COMPANY

EDITORIAL OFFICE — 1010 BEAUMONT BLDG., 8
BUSINESS OFFICE — 7200 WYDOWN BL., 5

ST. LOUIS, MO., U.S.A.

COPYRIGHT, 1945
ANNALS PUBLISHING COMPANY

Annual Subscription in United States, Spain, Central and South America, $6.00 in Advance. Canada, $6.40. Other Countries, $6.80.

RF 1
A613
v.54

OHIO STATE
UNIVERSITY

Table of Contents, Volume 54

MARCH, 1945—NUMBER 1

PAGE

I—Bronchogenic Carcinoma. An Analysis of 175 Proved Cases, Paul H. Holinger, M.D., H. James Hara, M.D., and Edwin F. Hirsch, M.D., Chicago, Ill. 5

II—Malignant Exophthalmos, LeRoy A. Schall, M.D., and Daniel J. Reagan, M.D., Boston, Mass. 37

III—Nasal Allergy For the Practicing Rhinologist, George E. Shambaugh, Jr., M.D., Chicago, Ill. 43

IV—Post-War Deafness, A Challenge to the Otologist, Bernard J. McMahon, M.D., St. Louis, Mo. 61

V—Aerosinusitis—A Résumé, Colonel Paul A. Campbell, M.C., Randolph Field, Texas 69

VI—Local Infiltration of Penicillin Solutions In Peritonsillar and Pharyngomaxillary Spaces, Major A. J. Cone, M.C., U. S. Army, Ellington Field, Texas 84

VII—An Adaptation of the Displacement Principle for the Introduction of Gases Into the Sinuses, Arthur W. Proetz, M.D., St. Louis, Mo. 91

VIII—Cilia and Penicillin, A Laboratory Note, Arthur W. Proetz, M.D., St. Louis, Mo. 94

IX—Brief Labyrinthine Physiology for Residents as Part of Bárány Instruction at the Massachusetts Eye and Ear Infirmary, Francis L. Weille, M.D., Boston, Mass. 96

X—Bronchography As A Diagnostic Aid In Chest Disease, George S. McReynolds, Jr., M.D., and Fred W. Shelton, M.D., Galveston, Texas 114

XI—Individualization in the Management of Carcinoma of the Maxillary Sinus, Maurice F. Snitman, M.D., Chicago, Ill. 125

XII—The Significance of Hoarseness, Waitman F. Zinn, M.D., Baltimore, Md. 136

XIII—Some Lesions Associated with Fistula of the Frontal Sinus, Harold L. Hickey, M.D., Denver, Colo. 143

730950

PAGE

XIV—Laryngeal Stroboscopy, H. B. Perlman, M.D., Chicago, Ill. 159

XV—Penicillin and the Infections of the Ear, Nose and Throat, M. H. Mothersill, M.D., Indianapolis, Ind. 166

XVI—Seven Charts on Cancer of the Larynx, Le Roy A. Schall, M.D., Boston, Mass. 174

XVII—The Alnico Magnet—An Aid to Bronchoscopy and Esophagoscopy, Murdock Equen, M.D., Atlanta, Ga. 178

XVIII—Hearing Testing Cabinet, J. Kent Leasure, M.D., Indianapolis, Ind. 183

XIX—Foreign Bodies of the Maxillary Sinus, John Clair Howard, Jr., M.D., Lieut., M.C., U.S.N.R., Kansas City, Mo. 186

XX—Microscopy of the Temporal Bone with Polarizing Films, George Kelemen, M.D., Boston, Mass. 192

Clinical Notes

XXI—Massive Pharyngeal Hemorrhages, George H. Woodruff, M.D., Joliet, Ill. 202

Society Proceedings

Sixty-Sixth Annual Congress of the American Laryngological Association, New York, N. Y., June 7-8, 1944. Stereoscopic Radiographs vs. Single Films—Laryngocele—Pathology of Nasal Mucous Membrane and Suggestions as to Treatment—Inter-American Relations in Otolaryngology—Some Problems in the Surgical Treatment of Bilateral Abductor Paralysis of the Larynx—Extralaryngeal Surgical Approach for Arytenoidectomy; Bilateral Abductor Paralysis of the Larynx—An Unusual Case of Carcinoma of the Larynx—Fibrosarcoma of the Larynx—Congenital Dermoid of the Pharynx—Neurofibroma of the Nasopharynx—Atresia of the Pharynx Following Tonsil and Adenoid Operation—Congenital Lymphangiomatous Macroglossia, with Cystic Hygroma of the Neck—Malignant Recurring Nasal Hemorrhage of Undetermined Etiology—Muscles of Phonation and Deglutition as Viewed Through the Operative Wound in Frontal and Ethmoid Sinus—Tumor of the Carotid Body 208

Chicago Laryngological and Otological Society, Meeting of Monday, November 6, 1944. Practice of Otolaryngology In China In Peace and War 219

Meeting of Monday, December 4, 1944. Massive Pharyngeal Hemorrhages: Case Report 221

Meeting of Monday, January, 8, 1945. Alterations In the Brain Due To Asphyxiation—The Therapeutic Value of Lipiodol In Sinus Infection 223

Obituaries

Sir James Dundas-Grant 235

Ira Frank 237

Books Received 240

Notices 241

Officers of the National Otolaryngological Societies 242

JUNE, 1945—NUMBER 2

PAGE

XXII—Disturbance of the Function of the Salivary Glands, A. C. Furstenberg, M.D., and Elizabeth Crosby, Ph.D., D.Sc., Ann Arbor, Mich. 243

XXIII—Chronic Suppurative Otitis Media, Lawrence R. Boies, M.D., Minneapolis, Minn., ... 265

XXIV—Plasma Cell Tumors of the Upper Part of the Respiratory Tract, Frederick A. Figi, M.D., Albert C. Broders, M.D., and Fred Z. Havens, M.D., Rochester, Minn. 283

XXV—Irrigation of the Maxillary Sinus By Way of the Middle Meatus, O. E. Van Alyea, M.D., Chicago, Ill. 298

XXVI—The Respiratory Function of the Larynx—Some Observations On Laryngeal Innervation. Preliminary Report, John A. Murtagh, M.D., Hanover, N. H. 307

XXVII—A Simple and Practical Procedure for Developing Esophageal Voice In the Laryngectomized Patient, E. Trible Gatewood, M.D., Richmond, Va. 322

XXVIII—A New Treatment for Esophageal Obstruction Due to Meat Impaction, John R. Richardson, M.D., Boston, Mass. 328

XXIX—Management of Chronic Middle Ear Suppuration with Fistula, M. Tamari, M.D., and L. Hirsch, M.D., Chicago, Ill. 349

XXX—Prevention of Deafness In Children, Russell M. Decker, M.D., Pasadena, Calif. 358

XXXI—The Stenger Malingering Test Made with the Audiometer, Lt. Comdr. Russell Fletcher, M.C., U.S.N.R., San Diego, Calif. 362

XXXII—External Otitis in the South Pacific, Lt. Comdr. Roy F. Nelson, M.C., V(S), U.S.N.R., Shoemaker, Calif. 367

XXXIII—Maxillary Sinusitis of Dental Origin and the Management of Antral Fistula, Lt. Colonel Ralph J. McQuiston, M.C., A.U.S., Indianapolis, Ind. 373

XXXIV—Arterial Spasm and Fat Metabolism; Their Relation To Certain Diseases and to Certain Members of the Vitamin B Complex, Grant Selfridge, M.D., San Francisco, Calif. 384

XXXV—Further Observations on the King Operation for Bilateral Abductor Paralysis, Lewis Francis Morrison, M.D., San Francisco, Calif. 390

XXXVI—Spasmodic Facial Neuralgia, Robert Lee Glass, M.D., Indianapolis, Ind. 409

PAGE

XXXVII—Hemangioma of the Ear—A New Method for the Control of Hemorrhage, O. Jason Dixon, M.D., Kansas City, Mo. 415

XXXVIII—Clinical Observations on the Palatine Tonsil in the Aged, George Kelemen, M.D., Boston, Mass. 421

Abstracts of Current Articles 436

Notices 441

Officers of the National Otolaryngological Societies 442

SEPTEMBER, 1945—NUMBER 3

XXXIX—Management of Chronic Sinus Disease—A Critical Analysis of Modern Therapeutic Measures. O. E. Van Alyea, M.D., Chicago, Ill. 443

XL—The Role of the Otologist in a Hearing Conservation Program. W. E. Grove, M.D., Milwaukee, Wis. 458

XLI Chronic Deafness, Its Treatment In the Army. Major Edward H. Truex, Jr., M.C., Butler, Pa. 466

XLII—Stroboscopic Examination of the Ear. H. B. Perlman, M.D., Chicago, Ill. 483

XLIII—Recurrent Severe Hemorrhage From the Nose and Pharynx. Francis B. Sargent, M.D., Providence, R. I. 495

XLIV—Aero-Otitis Media; A Further Report of Purulent Otitis Media Complicating Aero-Otitis Media. Major R. Wesley Wright, Medical Corps, U. S. Army Air Corps 499

XLV—Acoustic Trauma. Lilburn C. Boemer, Major, Medical Corps 513

XLVI—Cancellous Bone Transplants for the Correction of Saddle Nose. Samuel Fomon, M.D., New York, N. Y., Romeo Luongo, M.D., Philadelphia, Pa., Alfred Schattner, M.D., New York, N. Y., and Frank Turchik, M.D., Bridgeport, Conn. 518

XLVII—The Management of Fractures of the Nasal Bony Vault. Major Joseph H. Hersh, M.C., A.U.S., Camp Stoneman, Calif. 534

XLVIII—Rupture of the Tympanic Membrane Due to Blast—A Mechanical Hypothesis. J. J. Cantor, B.S., Los Angeles, Calif. 554

XLIX—Postbronchoscopic Reactions. Major L. L. Titche, M.C., A.U.S., Tucson, Ariz. 568

PAGE

L—Thornwaldt's Syndrome. A. R. Hollender, M.D., and Paul B. Szanto, M.D., Chicago, Ill. 575

LI—Ossifying Fibroma of the Ethmoid Cells and the Frontal Sinus—Report of a Case. George H. Woodruff, M.D., Joliet, Ill. 582

Society Proceedings

Chicago Laryngological and Otological Society, Meeting of Monday, February 5, 1945. Clinical Significance of Hoarseness—Irrigation of Maxillary Sinus by Way of Middle Meatus 586

Meeting of Monday, March 5, 1945. Visualization of Otitic Brain Abscess—Carcinoma of the Sphenoid Sinus—Temporal Lobe Syndrome Following Mastoidectomy—Ossifying Fibroma of the Ethmoid Cells and Frontal Sinus 598

Obituaries

Horace Newhart, M.D. 609

Robert Levy, M.D. 615

Abstracts of Current Articles 619

Hanau W. Loeb Memorial 635

Notices 646

DECEMBER, 1945—NUMBER 4

LII—The Use of Radium in the Aerotitis Control Program of the Army Air Forces 650

Irradiation Program in the First Air Force 661

LIII—Activities of the Irradiation Clinic At Westover Field, Massachusetts. Captain John E. Hendricks and Captain Alfred T. Lieberman, Medical Corps, Army of the United States 662

LIV—Report of 52 Cases of Recurrent Aerotitis; First Air Force, Mitchel Field, New York. Major Ernest A. Weymuller and Major Paul L. Magnuson, Medical Corps, Army of the United States 684

LV—Report of the Third Air Force Irradiation Unit, 31 August 1945. Captain Jerome J. Glauber, Major John W. Smith, Medical Corps, Army of the United States, and Lt. Colonel Donald H. Earl, United States Army 686

LVI—Aerotitis and Radium Therapy in the Eighth Air Force. Major Braswell E. Collins, Major John W. Eschenbrenner, Jr., and Captain Phillip L. Lyle, Army of the United States 694

PAGE

LVII—The Use of Radium in the Aerotitis Control Program in the Twelfth Air Force. Major John S. Mikell, Medical Corps, Army of the United States 708

LVIII—Aerotitis Program in the Fifteenth Air Force. Captain Tony J. Trapasso, Medical Corps, Army of the United States 716

LIX—Summary of the Combined Report 722

LX—Some Further Experiments in the Production of Negative Pressure in the Trachea and the Frontal Sinus by Ciliary Action. A. C. Hilding, M.D., Duluth, Minn. 725

LXI—"Postnasal Drip"—The Current American Nightmare. Arthur W. Proetz, M.D., St. Louis, Mo. 739

LXII—Nasal Psychosomatic Syndromes Accompanying and Following Acute Anxiety. Major Winfred L. Post, M.C., Army Air Forces 747

LXIII—The Use of Tantalum in Radical Frontal Sinus Surgery. Robert L. Goodale, M.D., Boston, Mass. 757

LXIV—Pneumosinus Dilatans. Hans Brunner, M.D., Irwin G. Spiesman, M.D., and John J. Theobald, M.D., Chicago, Ill. 763

LXV—Treatment of Suppurative Paranasal Sinusitis with Repeated Irrigations of Penicillin—A Negative Report. Major Robert E. Priest, Medical Corps, Army of the United States 786

LXVI—Photography Through A Nasopharyngoscope. Captain John E. Hendricks, Medical Corps, Army of the United States 797

LXVII—Ménière's Syndrome: The Validity of the Intradermal Histamine Test. Miles Atkinson, M.D., New York, N. Y. 801

LXVIII—Mixed Salivary Tumor in the Right Tonsil Fossa with Narcolepsy and Cataplexy. Charles E. H. Bates, M.D., San Francisco, Calif. 812

Obituary

Isidore Friesner, M.D. 819

Abstracts of Current Articles 821

Books Received 835

Notices 837

Officers of the National Otolaryngological Societies 838

Index of Authors 839

Index of Titles 841

Contents.

PAGE

LII—The Use of Radium in the Aerotitis Control Program of the Army Air Forces 650

Irradiation Program in the First Air Force 661

LIII—Activities of the Irradiation Clinic At Westover Field, Massachusetts. Captain John E. Hendricks and Captain Alfred T. Lieberman, Medical Corps, Army of the United States 662

LIV—Report of 52 Cases of Recurrent Aerotitis; First Air Force, Mitchel Field, New York. Major Ernest A. Weymuller and Major Paul L. Magnuson, Medical Corps, Army of the United States 684

LV—Report of the Third Air Force Irradiation Unit, 31 August 1945. Captain Jerome J. Glauber, Major John W. Smith, Medical Corps, Army of the United States, and Lt. Colonel Donald H. Earl, United States Army 686

LVI—Aerotitis and Radium Therapy in the Eighth Air Force. Major Braswell E. Collins, Major John W. Eschenbrenner, Jr., and Captain Phillip L. Lyle, Army of the United States 694

LVII—The Use of Radium in the Aerotitis Control Program in the Twelfth Air Force. Major John S. Mikell, Medical Corps, Army of the United States 708

LVIII—Aerotitis Program in the Fifteenth Air Force. Captain Tony J. Trapasso, Medical Corps, Army of the United States 716

LIX—Summary of the Combined Report 722

LX—Some Further Experiments in the Production of Negative Pressure in the Trachea and the Frontal Sinus by Ciliary Action. A. C. Hilding, M.D., Duluth, Minn. 725

LXI—"Postnasal Drip"—The Current American Nightmare. Arthur W. Proetz, M.D., St. Louis, Mo. 739

LXII—Nasal Psychosomatic Syndromes Accompanying and Following Acute Anxiety. Major Winfred L. Post, M.C., Army Air Forces 747

CONTENTS—Continued

PAGE

LXIII—The Use of Tantalum in Radical Frontal Sinus Surgery. Robert L. Goodale, M.D., Boston, Mass. 757

LXIV—Pneumosinus Dilatans. Hans Brunner, M.D., Irwin G. Spiesman, M.D., and John J. Theobald, M.D., Chicago, Ill. 763

LXV—Treatment of Suppurative Paranasal Sinusitis with Repeated Irrigations of Penicillin—A Negative Report. Major Robert E. Priest, Medical Corps, Army of the United States 786

LXVI—Photography Through A Nasopharyngoscope. Captain John E. Hendricks, Medical Corps, Army of the United States 797

LXVII—Ménière's Syndrome: The Validity of the Intradermal Histamine Test. Miles Atkinson, M.D., New York, N. Y. 801

LXVIII—Mixed Salivary Tumor in the Right Tonsil Fossa with Narcolepsy and Cataplexy. Charles E. H. Bates, M.D., San Francisco, Calif. 812

Obituary

Isidore Friesner, M.D. 819

Abstracts of Current Articles 821

Books Received 835

Notices 837

Officers of the National Otolaryngological Societies 838

Index of Authors 839

Index of Titles 841

ANNALS
OF
OTOLOGY, RHINOLOGY
AND
LARYNGOLOGY

VOL. 54 MARCH, 1945 No. 1

I

BRONCHOGENIC CARCINOMA:

AN ANALYSIS OF 175 PROVED CASES

PAUL H. HOLINGER, M.D.

H. JAMES HARA, M.D.

AND

EDWIN F. HIRSCH, M.D.

CHICAGO, ILL.

During the year 1940 carcinoma of the lung and pleura, according to the U. S. Government report,[1] was the cause of 5,556 deaths in the United States. Based on per 100,000 population the death rate was 1.1 in 1920 and 2.2 in 1930 and 4.0 in 1940.[1] Improved methods of diagnosis and a greater interest of the profession in this form of pulmonary pathology have contributed much toward its earlier recognition. It is generally conceded that there has been not only an apparent but also an actual numerical increase in this disease.[2] Bronchogenic carcinoma is fifth in frequency in types of carcinoma among males; it is surpassed only by carcinoma of the stomach, of the intestines and rectum, the prostate and bladder, and the liver and pancreas. According to some writers,[3] carcinoma of the lung is second only to that of the stomach and of the large intestine;

Presented before the Twenty-Sixth Annual Meeting of the American Broncho-Esophagological Association, New York, N. Y., June 6, 1944.

MEDICAL LIBRARY

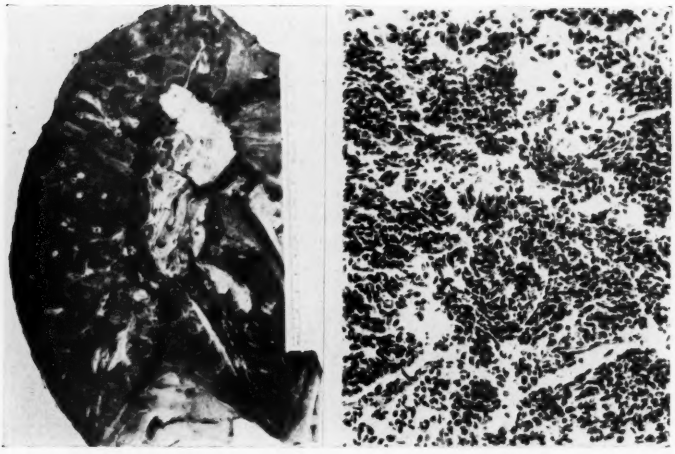


Fig. 1.—Hilar tumor (small-celled bronchogenic carcinoma) invading the left main bronchus of a lung with nodular silicosis. The small black regions are the silicosis nodules. 700 grams of moist lung contained 70.2 mg. of silica (SiO_2).

Oschsner and DeBakey[4] state that it constitutes 10 per cent of all deaths due to cancer.

The present study is concerned with 175 proved cases of primary bronchogenic carcinoma observed principally at the Research and Educational Hospitals of the University of Illinois and at St. Luke's Hospital, Chicago, prior to February, 1944. Isolated cases were examined in other institutions. The majority of these patients were admitted to one of these institutions for the purpose of diagnosis.

ETIOLOGY

As in all other cancerous lesions in the body, no primary cause is known. Innumerable secondary factors have been suggested and scrutinized. In an exhaustive review of the literature Simons[5] mentions no less than 14 different predisposing causes. These are discussed further by Loizaga.[6] It is generally agreed that no single agent is the sole cause, but that a chronic irritant is an essential common factor. Certain occupations seem to predispose to cancer of the lung. A notable example is found among the miners in the district of

Schneeberg, Saxony.[5] It is reasonably well established that the high incidence of bronchogenic carcinoma in this group is related to the radioactivity of the inhaled dust. In our series, one patient with bronchogenic carcinoma, proved by needle aspiration after bronchoscopy failed to demonstrate the suspected tumor, was shown postmortem to have silicosis as well (Fig. 1). It is impossible to conclude an etiologic relationship from this single case. Others[7, 8] have reported similar isolated cases, but Vorwald and Karr[9] found that in a significant number of autopsies the incidence of carcinoma of the lung in silicotic persons was no higher than that in non-silicotic persons.

Exhaust gases from automobiles and tar on roads have been considered as etiologic factors. Two of our patients, husband and wife, travelled extensively from one job to another by automobile. He was a road builder. Both died of bronchogenic carcinoma. Without further corroborative evidence, this, too, must be considered a coincidental finding.

The inhalation of tobacco in smoking is considered by some as a factor causing cancer. It is generally known that cancer among smokers is much more prevalent in the so-called smoke track—the lips, mouth, larynx and bronchi—than in nonsmokers. Ninety per cent of a large series of patients observed at the Cook County Hospital were smokers.[10] Fifty of our own patients were heavy smokers. However, 14 stated that they did not smoke, and there is no mention of this habit in the remainder of the histories.

Other pulmonary diseases such as influenza, bronchitis and bronchiectasis, tuberculosis, and trauma have been suggested as etiologic factors of bronchogenic carcinoma. It is of particular interest in this connection that as Wells[11] stated in 1927, if the increase in bronchogenic carcinoma then already apparent is due to the peculiar hyperplasia associated with the repair of the epithelium destroyed in the influenza epidemic of 1918 and 1919, then a decrease in the incidence of the disease should follow. As is evident, this has not followed.

Age, Sex and Race Distribution (Fig. 2): Bronchogenic carcinoma is more frequently observed in white males. Ninety-one per cent of 111 patients observed by Stephens[12] and 75 per cent of 536 patients reported by Singer[13] were males. Perrone and Levison[14] stated that 34 of 38 of their patients who came to autopsy and 61 of 77 who were examined bronchoscopically were males. In our group there were 150 white males, one negro and one Filipino, and 23 white

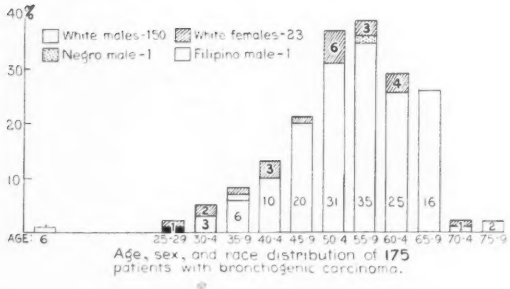


Fig. 2.—Age, sex, and race distribution.

females. Thus the proportion of our series is 87 per cent male and 13 per cent female.

It is of interest to note that carcinoma of the bronchus may appear at any age. Holman[15] quoted a number of authors who reported cases of carcinoma of the lung among children. According to the United States Government's vital statistics,[1] in 1940 one death from this disease was reported in an infant under one year of age; 3 under 2 years; 2 under 4 years; 8 between 5 and 9 years; 18 between 10 and 18; 22 between 15 and 19; 44 between 20 and 24; and 38 between 25 and 29 years of age. In our series the youngest patient was a six-year-old Italian girl. The oldest was a 76-year-old man. Bronchogenic carcinoma occurs most frequently after 40 years of age. Forty-three per cent of the cases in our series were in the single decade of 50-59 years and 72 per cent were between the ages of 45 and 64.

The negro population constitutes about 10 per cent of the population of the United States. In the city of New Orleans it constitutes about 50 per cent. Halpert[16] reported that in a ten-year survey of necropsies in the Charity Hospital at New Orleans 3,838 white and 5,024 negro subjects came to autopsy, a proportion of 4:5. Carcinoma of the lung was found in 81 whites and 54 negroes, a proportion of 1½:1. In the total necropsy material there were 5,635 males (2,610 white and 3,025 negro males) and 3,227 females (1,228 white and 1,999 negro females), a proportion of almost 2:1.

Among 2781 autopsies at the University of Chicago Quinland[17] encountered 74 with carcinoma of the lung. Only 3 of these were in negroes. (Fig. 2)

PATHOLOGY

According to opinions now prevailing, practically all carcinomas of the lung arise in bronchial tissue. This generalizing statement does not disregard reports of cancerous epithelial growths believed to have originated in the alveolar tissues of the lung. A large number of the primary carcinomas of the lung appear at or near the hilum, growing from the main bronchus or its primary divisions; others occur in the substance of the lung or near the pleural surface. Bronchogenic carcinoma shows a distinct predilection for the right bronchus. This is generally attributed to the more direct course and greater diameter of the right bronchus than the left, thus being exposed to a greater irritation, as well as to the greater total surface area of the right bronchus. In 111 of our patients the neoplasm occurred on the right and in 66 on the left; it was bilateral in 4 (Fig. 3). The proportion is 11:6 in favor of the right bronchus. Fifty-five of the tumors on the right side were found in the primary bronchus within 1-2 cm. of the carina, 19 at the orifice of the upper lobe bronchus, 2 at the orifice of the middle lobe and 32 in the lower lobe bronchus. Of the 60 growths originating on the left side, 20 were in the primary bronchus, 15 at the opening of the upper lobe and 23 in some portion of the lower lobe bronchus. In 4 cases the tumor apparently had originated near the bifurcation and involved both bronchi. Peripheral growths were found in 3 cases on the right and 2 on the left. In endobronchial growth it will be recalled that only the proximal portion of the growth is observed upon bronchoscopy. Thus in many instances the exact site of origin could not be determined. This was particularly true in advanced lesions in which the examining tube failed to pass beyond the obstructing neoplasm.

The size of bronchogenic carcinoma of the lung varies greatly. The bulky tumors, in roentgen films, cast large shadows in the lung fields. The bronchus from which such a large growth develops is like a stalk, capped by the carcinoma tissues (Fig. 4). Portions of these bulky tumors become necrotic and liquefy. Erosion of blood vessels by these changes and tumorous tissue growth cause hemorrhage, occasionally severe.

The more common variety of bronchogenic carcinoma occurs at or near the hilum. It is a firm grey tissue mass usually limited in size between 1 to 6 cm. and with marginal fibrous radiations into the lung parenchyma. The lumen of the associated bronchus is constricted, the longitudinal folds of the lining are replaced by granular or nodular tissues, and the thickened bronchial wall merges into

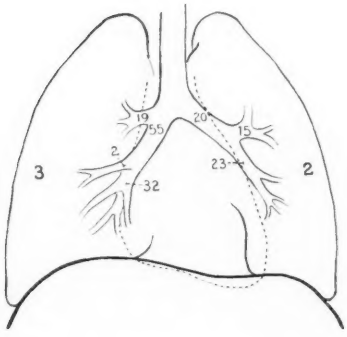


Fig. 3.—Distribution of 175 cases of primary bronchogenic carcinoma. 111, or 63%, were found in the right lung; 60, or 34%, in the left; and 4, or 3%, were bilateral. Three were found in the periphery of the right lung, two in the left.

the tumor mass (Fig. 5). Another form of bronchogenic carcinoma that easily escapes detection in gross examinations of the lungs is the small warty tumor which Kikuth[18] mentioned in his report. These appear usually near the origin of the bronchi leading to the individual lobes of the lung. The bronchial mucosa has a rough thickening or scarcely elevated tissues only a few millimeters in size. The lymph channels of lungs with these tumors are invaded extensively by carcinoma so that peribronchial tissues of the entire lung, one lobe especially, have diffuse or focal fibrous tissue thickenings and the pleura has many single and confluent grey fibrous plaques (Fig. 6). Among these tumors are some where the site of the primary focus may not be established definitely and the lungs show a diffuse thickening of the peribronchial tissues.[19] Histological examinations of these lungs demonstrate a diffuse infiltration of the peribronchial lymph channels and tissues by carcinoma cells associated with growths of fibroplastic stroma (Fig. 7). The air passages at the site of the tumor are constricted, the lumina beyond become dilated and filled with retained secretion. Complications of pneumonia or even abscesses occur in these portions of the lungs.

Small papillary or polypoid carcinomas in the parenchyma of the lungs cast no shadows in roentgen films. Their metastases produce focal symptoms in viscera other than the lungs as well as gen-

Fig. 4.—Large, bulky tumor of the right bronchus with extensive necrosis.

eralized systemic manifestations. The invasion of the pulmonary blood vessels by carcinoma facilitates the systemic distribution of the disease.

Hilar carcinomas of the lung, spreading by extension and metastasis into the mediastinum, compress the pulmonary blood vessels or encroach upon the heart. Compression of the pulmonary arteries or veins impedes the circulation of the blood in the lungs, which in some patients seems to be the main cause of their dyspnea.

The growth of the carcinoma cells into the lymph channels and their invasion of the walls of the pulmonary blood vessels in the lungs provide means for widespread dissemination of the disease into many tissues of the body. Metastatic carcinoma of the pleura and the hilar lymph nodes through the lymph channels is common. Metastases appear in other mediastinal and cervical lymph nodes, and by their enlargement or extensions produce other complications. Secondary growths in the pleura, brain, bones, kidneys, suprarenal glands, skin, liver, thyroid gland, myocardium and other places not infrequently cause symptoms which demonstrate the clinical course of the disease, while those due to the tumor growth in the lungs are insignificant. In many of these patients the bizarre character of the clinical symptoms aroused by the metastases creates an enigmatic diagnostic problem. Should a metastatic lesion approachable by biopsy procedures

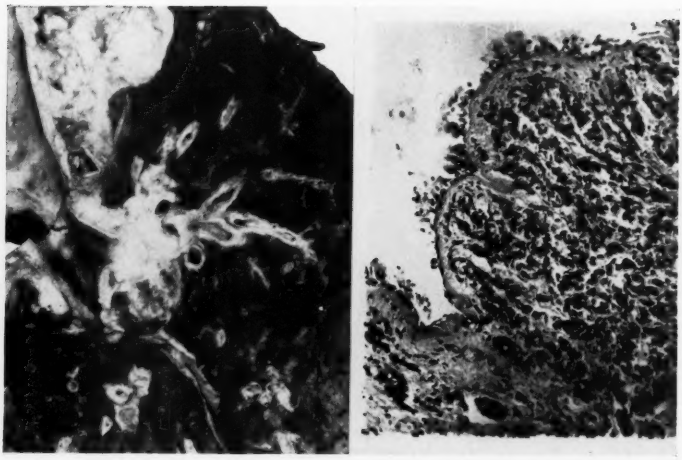


Fig. 5.—Hilar carcinoma of the right bronchus radiating into the right upper lobe to simulate tuberculosis. The small carcinoma cells of these tumors penetrate the tunica propria of the mucosa and extend along the peribronchial lymph channels to the pleura. The primary growth causes a granular roughening of the lung, a thickening of the bronchial wall, and a stenosis of the lumen.

be discovered and examined during life, a clue to the nature of the disease is obtained. Otherwise, the carcinoma is revealed only by a careful postmortem examination.

The histologic structure of carcinomas of the lungs varies through a considerable range both as regards the character of the individual cells and their arrangement. Probably the commonest variety is the form composed of pavement epithelial cells and described as squamous cell. These tumors contain cells which simulate those of various layers of a mucosal squamous epithelial surface. The cells, usually large, are atypical in size, shape, arrangement, and in the amount of hornification that occurs (Figs. 8 and 9).

At the opposite extreme in size is the variety composed of small epithelial cells that resemble those in the deeper portions of pseudostratified epithelium and are arranged in small alveolar masses. The individual cells have a darkly stained granular cytoplasm, scanty or more abundant, and a vesicular darkly stained nucleus with several coarse chromatin granules. In the histological preparations, tumors composed of these small cells may be confused with tissues of lym-

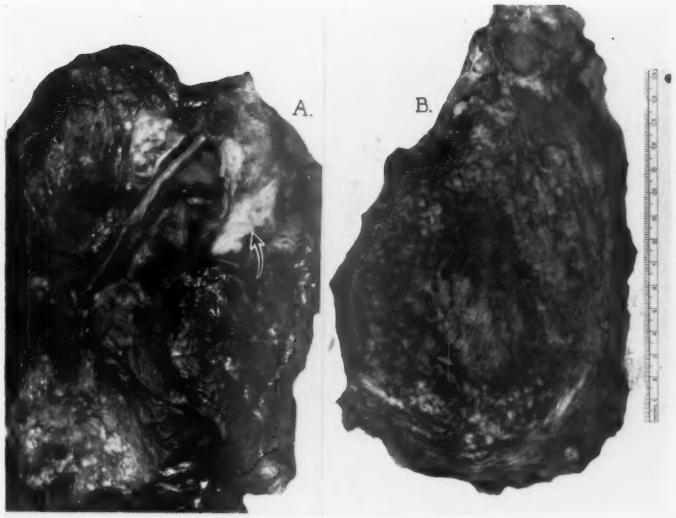


Fig. 6.—(a) Photograph illustrating an extramural carcinoma of the right main bronchus. (b) Extensive metastases covered the visceral and parietal pleura on both sides.

phocytic structure (Fig. 10). Carcinomas of this kind occur, which, while clearly not lymphocytic, contain markedly cellular tissues composed of small cells with little of the epithelial cell characteristics.

Other carcinomas contain large or small epithelial cells arranged in tubules or acini, and accordingly are designated as grandular on the basis of their histologic arrangement (Fig. 11). Occasionally microscopic preparations reveal that the cells of a carcinoma have a signet ring form, (Fig. 7) the cytoplasm containing a large vacuole with pseudomucin and a compressed nucleus displaced to the cell membrane. This formation of vacuoles is simulated also in certain tissues where rather large aggregates, arranged in mosaics, have scattered and lumen-like structures, also containing a pseudomucin.

Tissues taken for biopsy successful in revealing the presence of carcinoma are the important elements in establishing a diagnosis. Many of the primary tumors because of their position near the hilum of the lung can be reached with instruments through the bronchoscope. The pieces of tissue removed from the bronchial lining and wall for histological examination are usually small, but if representa-

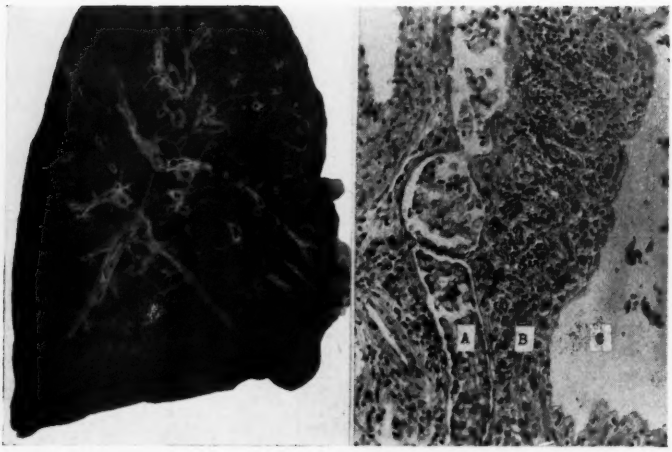


Fig. 7.—Diffuse carcinomatous invasion of the bronchial walls. The primary focus could not be established. In the photograph of the surface made by hemisecting the inflated formalin-fixed right lung, the marked fibrous thickening of the bronchial walls can be seen. The masses of tumor cells spreading along the peribronchial lymphatics are illustrated in the photomicrograph. (a) Lymphatic channel invaded by tumor cells having a signet ring form. (b) Bronchial wall. (c) Bronchial lumen.

tive present no serious problem in tissue diagnosis. Inability to obtain representative tissues, whatever the reason, complicates this direct process of diagnosis. Another factor, which apparently is dependent upon the construction of the biopsy instruments, is the distortion or crushing of the tissues during removal so that the details of cell structure are destroyed. This occurs especially with tumor tissues composed of small cells. These, moreover, are the forms which must be distinguished from lymphoid tissue cells and which demand histological preparations without faults. Sometimes fragments of tumor tissue acceptable for diagnostic purposes are obtained by bronchial aspiration. Frequently these are mixed with blood cells.

Carcinoma, metastatic in the various accessible viscera, may be revealed also by biopsy procedures on these organs. On the basis of this material the presence of a primary focus in the lungs of a patient can be inferred only to the extent that cell characteristics in the biopsy tissues or the clinical data indicate. The examination of aspirated bronchial secretions and pleural exudates in sections of their sediments affords another approach in obtaining data for the clinical

diagnosis of carcinoma of the lungs (Fig. 12). The results so obtained sometimes have surprising value.

The unusual clinical symptoms caused in patients by bronchogenic carcinoma metastases of bones have aroused comment. Some of these experiences in which the symptoms and clinical observations in patients were confusing have been reported. In a patient observed clinically at St. Luke's Hospital multiple carcinoma metastases of the thyroid resulted in symptoms for which a thyroidectomy was done. The postmortem examination shortly thereafter demonstrated a primary small-celled bronchogenic carcinoma of the lung and metastases in many other viscera. The febrile disease of another patient in this hospital was studied carefully by physical examination and many laboratory tests including those for demonstrating typhoid, paratyphoid and malta fever infections. A clue to the nature of the disorder was provided by the biopsies of two skin specimens, one from the wrist, the other from the forehead. Each had a metastatic nodule of carcinoma 2 mm. in diameter in the corium tissues. The primary focus in the lungs seemed probable because of the character of the cells in the metastases, although careful physical and roentgen examinations failed to demonstrate a tumor in the lungs. Later, clinical manifestations of cerebral metastases appeared and the postmortem examination disclosed a small polypoïd carcinoma of the parenchyma of the lung, midway between the hilum and the pleural surface. This nodule and its polypoid extension into a branch of the pulmonary artery was only 2 x 2 x 4 cm. (Fig. 13). In another patient, a hilar carcinoma of the right lung invaded the pulmonary artery causing obturator thrombosis with necrosis and subsequent liquefaction of the entire lung, except for a few hilar structures (Fig. 14). The development of bronchiectasis (Fig. 15) and necrosis (Fig. 16) of the lung tissue beyond the tumor frequently occurs.

These specific instances of unusual sequelae in patients with bronchogenic carcinoma of the lungs by no means cover the range of possibilities. They serve mainly to emphasize that bronchogenic carcinomas of the lungs or their metastases may produce a great variety of clinical symptoms besides those commonly described.

SYMPTOMS

Initial Symptoms: The onset of bronchogenic carcinoma is almost always insidious. A number of our patients were unable to state the exact date of onset of symptoms. Almost all the patients complained of disorders referable to the respiratory tract (Fig. 17). Cough was the initial symptom in 50. The cough was slightly irri-

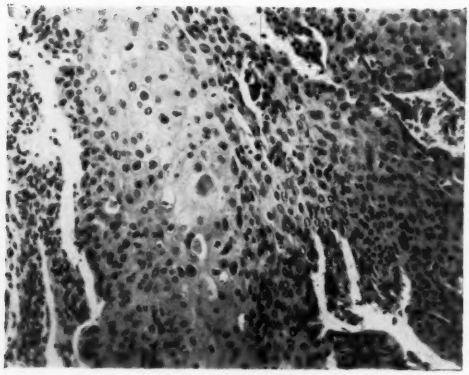


Fig. 8.—Photomicrograph illustrating a hornified squamous cell carcinoma of the bronchus. (x118)

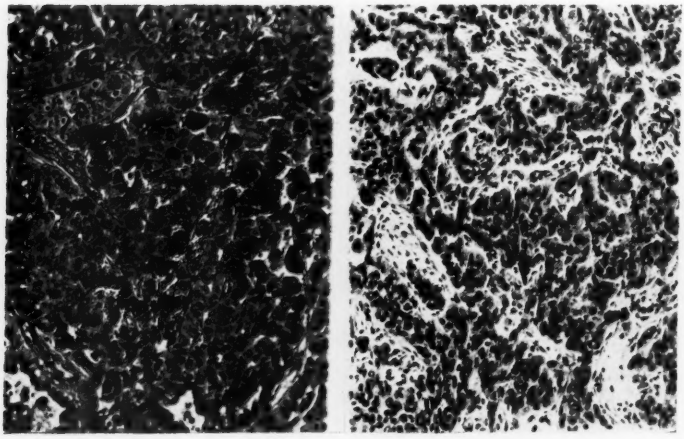


Fig. 9.—Photomicrographs illustrating two forms of squamous cell carcinoma without hornification. (x107)

tating and in its initial phase was always nonproductive. Later it became more productive of mucoid secretion. A number of smokers attributed their coughs to the irritating effect of tobacco on the respiratory mucous membrane and failed to realize a change in the character of their cough quite obvious to the family. Thirty-eight patients in this series stated that their trouble began with what they termed a cold or bronchitis but that unlike their usual colds, lasting a week or ten days, it stayed much longer. They accepted it as of no significance and did not seek medical attention early. Others thought that they had repeated colds at short intervals of two to four weeks, each attack lasting longer and leaving them much weaker than the previous one. Some had returned to their former occupations only to find the work too strenuous to continue after a trial of varied duration. Several described their early symptoms as a "spring cold", "summer cold", or "winter cold", depending on the season during which the symptoms appeared.

In an early stage bronchogenic carcinoma may simulate pulmonary tuberculosis. Twenty patients of this group had been admitted to sanatoria for tuberculosis, receiving thorough methodical diagnostic studies, with negative findings for active tuberculosis. Some of these patients remained in sanatoria for six weeks to six months before bronchoscopic examinations were made. Those with healed tuberculosis presented an additional problem in diagnosis.

Pain in the chest was recorded in 14 patients as their initial symptom. The thoracic pain was always on the side affected, intermittent and of short duration. With the progress of the disease the pain became more pronounced and almost continuous, and was always worse on coughing. Pain associated with early bronchogenic carcinoma was usually not severe, but rather a sensation of tightness, possibly from associated atelectasis. This sensation was variously described as a feeling of constriction, binding around the chest and inability to fill the lung on deep inspiration.

At the onset a diagnosis of pneumonia was made in 14 patients and of "flu" in 12. One of the patients in this group had recurrent attacks of so-called "flu-pneumonia". So masking were the chest symptoms that the attending physician thought that the presence of faucial tonsils might have been a factor in reducing the patient's resistance to the upper respiratory infections. A tonsillectomy and an adenoidectomy were performed under general anesthesia only three months before the true nature of the thoracic pathology was recognized by an examination of tissues taken bronchoscopically. Pleurisy

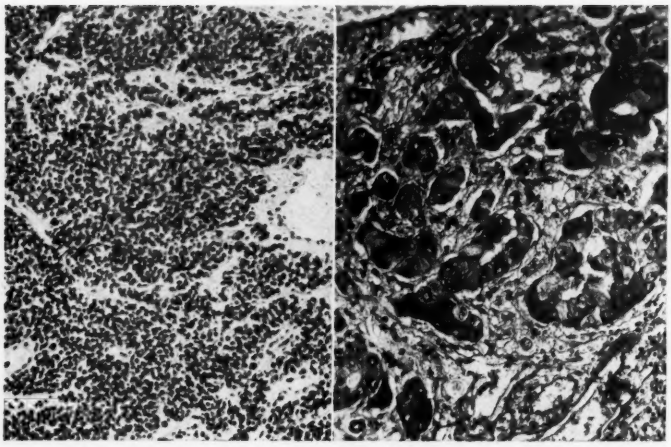


Fig. 10.—Photomicrograph illustrating a common form of small-celled bronchogenic carcinoma. (x107) The cellularity and soft character of these tumors render them susceptible to distortion by the trauma of the biopsy instrument.

Fig. 11.—Photomicrograph illustrating a glandular or adeno-carcinoma of the bronchus. (x107)

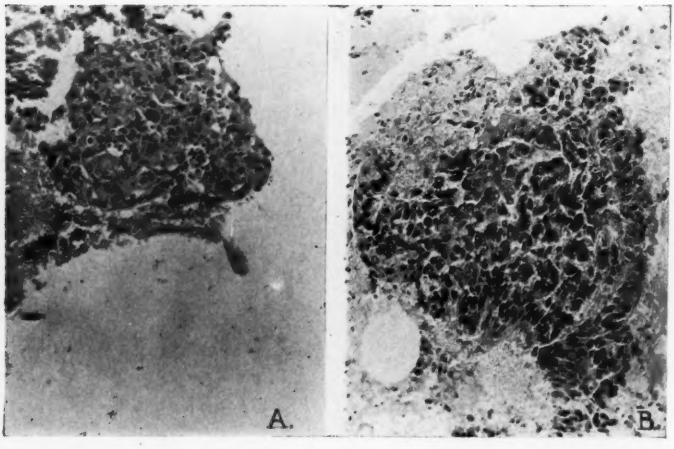


Fig. 12.—Photomicrographs illustrating carcinous tissues obtained by (a) bronchoscopic aspiration and (b) thoracentesis. They demonstrated the presence of a bronchogenic carcinoma in patients in whom the tumor was not accessible to the bronchoscope.

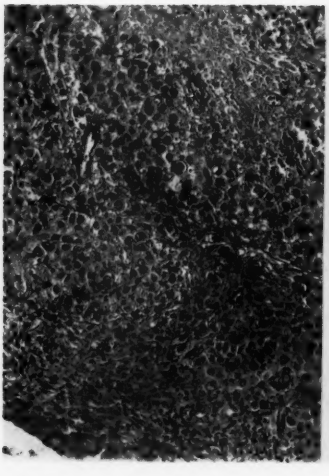
was given as the initial symptom in 8 patients, alteration in quality of voice in 4, and hemoptysis of varying quantity in 4.

Two of our patients came from a psychopathic hospital and were too irrational to give their clinical histories. One had no symptom referable to the chest. During the course of a routine physical examination, an x-ray film of the chest indicated the presence of a neoplasm. Subsequently, he developed metastases to the brain and gastro-intestinal tract, and death occurred two months after the original examination.

Symptomatology on Admission. Figure 18 indicates the outstanding symptoms in order of frequency which were recorded on admission to the hospital. These symptoms and their clinical implications are well known and one needs but touch on their salient features. Early in the course of this disease there is no group of symptoms, either subjective or objective, that is pathognomonic. The clinical picture presented depends on the size and the site of the growth, the stage and speed of its development, the presence or absence of metastases, the secondary inflammatory changes and the general condition of the patient. In the main, these symptoms are due to (1) pressure and obstruction within the bronchi and the lung parenchyma in the immediate vicinity of the growth, and (2) to the metastatic processes away from the original site of the tumor. Fully 98 per cent of the symptoms are referred to the chest. Cough, thoracic pain, expectoration and dyspnea are the four cardinal symptoms of carcinoma of the lung.

The most significant is the cough. One hundred fifty-three patients or 87 per cent complained of this symptom. Its onset is very early in the disease and it remains throughout the entire clinical course. It is usually progressive and fails to respond to rest or medication. It is irritating, dry and spasmodic at first, later becoming productive of clear, watery mucus, which soon turns purulent.

Thoracic pain was recorded in 122 patients or 69 per cent on admission. As previously stated, it is always on the side affected, and is described as a sensation of a band-like constriction around the chest. In some it appears as a substernal ache. This is described as being boring, stabbing or lancinating. In the earlier stages pain is always intermittent and of short duration. Later it becomes more continuous and severe. In some instances it is referred to the neck, shoulder and arm on the affected side. In patients with a peripheral growth, pain appears early. Severe thoracic pain develops upon in-



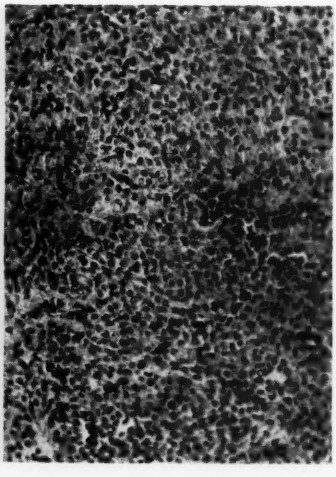


Fig. 13.—(a) Small papillary bronchogenic carcinoma of the right upper lobe. There were no pulmonary symptoms. The diagnosis of a carcinomatous disease was established by biopsies of nodules in the subcutaneous tissues. (b) Left forearm. (c) Right eyebrow. A tumor of the lung was considered the primary focus from the cell structure of the metastases. The postmortem examination revealed the primary pulmonary tumor and metastatic carcinoma of the brain, subcutaneous tissues of the head, neck, trunk, extremities and elsewhere.

vasion of the mediastinal, diaphragmatic or thoracic parietal pleura, and is an evidence of metastatic spread. Nineteen of our patients had this form of intense pleural pain. Much pain was recorded from metastases in the chest wall, rib erosion, intercostal neuritis, and brachial neuritis.

Dyspnea was recorded in 96 patients, or 55 per cent. A sudden development of dyspnea is an indication of acute atelectasis or spontaneous pneumothorax. It generally appears gradually, however, and gets progressively worse. When the tumor infiltrates a large portion of the lung parenchyma or when it blocks off a large major bronchus, the distal portion of the lung becomes atelectatic, resulting in anoxemia of varying degrees. Dyspnea also occurs in the presence of pleural effusion, pressure on the trachea from mediastinal metastasis or when the contralateral lung is invaded. There were four such instances of bilateral invasion. In those with cardiac insufficiency the symptoms originating from the heart at times overshadow those caused by the pulmonary neoplasm. Blood-streaked sputum in an individual past 40 years of age, who has previously been well and has developed a cough should arouse the suspicion of a pulmonary neoplasm. Its repeated occurrence is indicative of ulceration of the growth. Purulent expectoration indicates secondary infection, bronchiectasis or pulmonary abscess. Hemoptysis was recorded in 47 per cent of cases. These were not of an alarming quantity but were indicative of an extensive ulceration of an erosion of the larger vessels. Loss of weight, weakness, anorexia, emaciation, and cyanosis come late in the disease. Many patients retain their general health until sepsis begins. Fever above 100° F. is evidence of secondary infection from associated atelectasis, pneumonitis, bronchiectasis and abscess. Night sweats are considered a sign of general weakness and sepsis. Superior vena cava and Horner's syndromes appear very late in the disease, depending on the sites of the metastases.

Asthmatoid wheeze was recorded in 24 patients. It is always elicited in the presence of partial obstruction. Hoarseness was complained of by 21, and was caused by recurrent laryngeal nerve paralysis. Dysphagia occurs either from direct extension or secondary endo-esophageal growth. Vomiting is usually an indication of involvement of the liver and retroperitoneal nodes; it may occur in brain metastasis.

At times symptoms from metastatic growths overshadow those from the primary neoplasm and tax to the limit the diagnostic acumen of the most experienced. A chain of the most bizarre and confused

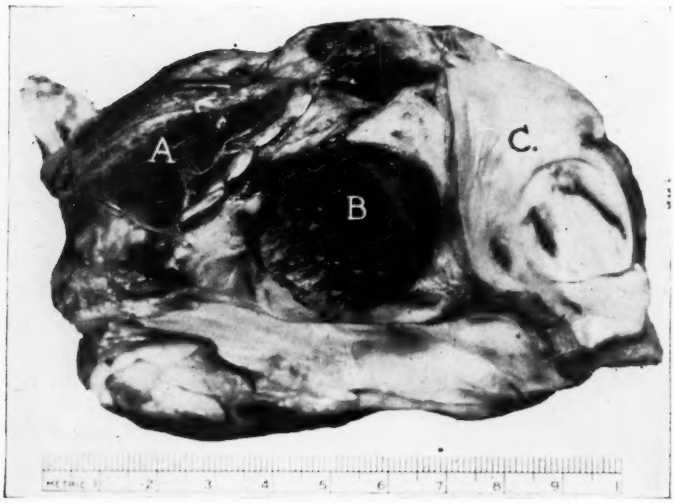


Fig. 14.—Tumor tissue and blood clot thrombosis of the right main branch of the pulmonary artery complicated this hilar bronchogenic carcinoma. The entire right lung, except the rigid structures at the hilum, was liquefied. 1400 cc. of a black fluid had been removed from the right pleura during life and 2080 gms. more were present at death. (a) Right bronchus. (b) Carcinomatous thrombosis of right branch of the pulmonary artery. (c) Pulmonary vein.

Fig. 15.—Gross features of a hilar (pavement cell) carcinoma of the left main bronchus. Bronchiectasis developed beyond the level of the tumor.

symptoms was recorded in a number of instances, leading to an erroneous diagnosis until very late in the disease. Examples of these startling cases have been previously reported,[19] and others are recorded in the discussion of the pathology.

PHYSICAL SIGNS

Physical signs are usually absent in the early stages of bronchogenic carcinoma. A small, peripherally located growth may remain silent until metastases occur. Signs of tumors in the stem bronchi are due to bronchial obstruction, ulceration, infiltration and secondary infection. Intrabronchial growths produce signs identical to those associated with bronchial foreign bodies, the dynamics of which have been so ably described by Jackson and Jackson.[20] Thus an obstruction may be partial or complete. Emphysema, either obstructive or compensatory, an asthmatoid wheeze, lobar pneumonia, localized pneumonitis, atelectasis, pleural effusion or any other abnormal chest sign should call for thorough investigation. Ipsilateral deviation of the trachea, a retraction of the chest, limitation of expansion of the affected hemithorax, dullness and flatness on percussion, elevation of the diaphragm, and suppressed or absent breath sounds are commonly found when obstruction is complete. Palpable supraclavicular, cervical or axillary nodes on the affected side are suggestive of metastases. A hemorrhagic pleural effusion, evidences of bone, brain, liver and other metastases appear late and make the diagnosis definite. Paralysis of the diaphragm or of one or both vocal cords is indicative of pressure upon, or direct involvement of, the nerve fibers by the growth. A Horner's syndrome may be observed late in the so-called superior sulcus tumor of Pancoast due to infiltration of the cervical sympathetic nerve or upper thoracic ganglion. The blood picture is not characteristic. It aids in the differentiation which must be made from infectious diseases of the lung such as tuberculosis, bronchiectasis, pneumonia, various virus diseases, lung abscess, and mycotic infections, and from such other conditions as Hodgkin's disease and lymphoblastoma.

ROENTGENOLOGY

The roentgen findings in this series of 175 cases of bronchogenic carcinoma have been discussed by Hartung and Wachowski[21] and Jenkinson and Hunter,[22] roentgenologists of the two institutions in which the majority of the patients of this series were studied. The principal feature roentgenologically is the extreme variation of the findings. No single finding or group of findings can be considered

Fig. 16.—Bronchogenic carcinoma surrounding and compressing the left main bronchus without invading its lumen. The surface made by cutting the lung demonstrates necrosis of the lung tissues beyond the tumor.

pathognomonic of the roentgen appearance of carcinoma of the bronchus. On the other hand, the diagnosis of bronchogenic carcinoma was considered in the interpretation of the roentgen study of such a large percentage of the cases that the x-ray examination must be considered to be the most accurate of the procedures which lead to a presumptive diagnosis of this disease.

The roentgen study of an individual suspected of having pulmonary pathology cannot be obtained through the interpretation of a single flat plate of the chest. A careful fluoroscopy, postero-anterior and lateral films, over-exposed films, the comparison of films taken during inspiration and expiration, occasional oblique views or stereoscopic sets are all essential features of diagnosis in certain instances of questionable pathology. Special diagnostic procedures consist of planography[23] and bronchography,[24] each of which may outline intra- or extra-bronchial tumors or may give evidence confirming a suspected bronchial obstruction.

X-ray findings per se are exceedingly variable. An early tumor in a small peripheral bronchus frequently manifests itself only by an area of localized "pneumonitis" which appears entirely inflammatory in character. When such a lesion clears and then recurs, the probability that it is due to a neoplasm is great. Areas of pulmonary suppuration in older individuals, or otherwise unexplained pulmonary suppuration in a patient of any age, must likewise be included in the group of lesions which, from the roentgenologic standpoint, may be due to pulmonary neoplasms. Thus a bronchus blocked by a tumor may become bronchiectatic, in which case the bronchiectasis may be the outstanding x-ray finding; or the center of a solitary round tumor may become necrotic and evacuate itself to reveal a round shadow with the characteristic fluid level of a pulmonary abscess.

The classical roentgen findings of bronchogenic carcinoma are those produced by varying degrees of bronchial obstruction.[25] Small tumors on the wall of a bronchus produce no changes in most instances and in such instances the negative x-ray findings in a patient who has occasional slight hemoptysis or who complains of a wheeze should not exclude further diagnostic studies, especially a bronchoscopic examination. Slightly larger tumors will manifest their presence on x-ray films by signs of obstructive emphysema, the heart and mediastinum being shifted away from the affected side on expiration; the over-inflated lung on expiration confirms the findings of the physical examination. Finally, total bronchial obstruction with

Fig. 17.—Initial symptoms of 175 cases of bronchogenic carcinoma.

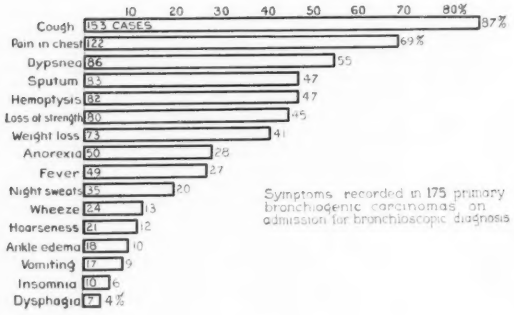

10 20 30 40 50 60 70 80%
Cough 153 CASES 87%
Pain in chest 122 69%
Dypsnea 86 55
Sputum 83 47
Hemoptysis 82 47
Loss of strength 80 45
Weight loss 73 41
Anorexia 50 28
Fever 49 27
Night sweats 35 20
Wheeze 24 13
Hoarseness 21 12
Ankle edema 18 10
Vomiting 17 9
Insomnia 10 6
Dysphagia 7 4%
Symptoms recorded in 175 primary bronchiogenic carcinomas on admission for bronchiloscopic diagnosis

Fig. 18.—Symptoms on admission to the hospital.

collapse of the segment, the lobe, or the whole lung distal to the tumor is the most common roentgenologic manifestation of an intrabronchial neoplasm.

BRONCHOSCOPY

A patient suspected of having bronchogenic carcinoma is examined bronchoscopically to permit removal of tissue for histologic study and to note various criteria which will aid in determining the operability or inoperability of the tumor.[26] A bronchoscopic examination should be made in patients with obscure pulmonary pathology as suggested by the history, symptomatology, physical and x-ray findings noted in the foregoing discussions. Such symptoms as a persistent cough, a wheeze, an unexplained hemoptysis, a recurring or so-called unresolved pneumonia are all specific indications for bronchoscopy. However, all too frequently such indications are ignored and this examination is postponed or not considered. The presumptive diagnosis of "atypical pneumonia" and the prolonged ineffective use of chemotherapy in an effort to clear a supposed pneumonia have been responsible for many delays in bronchoscopic examinations. In this series, six months or longer elapsed between the initial symptoms and bronchoscopy in 86 or approximately 50 per cent of the patients. In 14, or 8 per cent, of the patients, more than one year elapsed (Table 1). Among the latter group were three who had had a cough for more than ten years and could not accurately tell when their illness began.

TABLE 1.—TIME BETWEEN INITIAL SYMPTOMS AND BRONCHOSCOPY

14 cases (8%)	more than one year
72 cases (42%)	six months to one year
89 cases (50%)	less than six months

The bronchoscopic picture is extremely variable. Most frequently the carcinoma is a soft, red, fungating papillomatous type of tumor wich completely occludes a major bronchus. It may be covered with exudate, necrotic debris, or purulent material which must be carefully removed before tissue is taken. Such lesions are friable and bleed readily on contact with the lip of the bronchoscope, the aspirator or the forceps. Other tumors are polypoid in character with a mulberry-like appearance. Some carcinomas are invasive, pro-

ducing an extreme induration of the bronchial walls far above the point of the complete bronchial obstruction which was determined as the location of the lesion by physical and x-ray findings. Such lesions may cause bronchial stenoses high in the bronchus, with the actual intrabronchial invasion of the tumor far in the periphery, hiding the lesion which is characteristic of the picture of bronchogenic carcinoma from the bronchoscopic view. Occasionally an ulcerative type of lesion is found with rounded edges and a necrotic base extending along the floor or wall of the bronchus. Such lesions are not often seen bronchoscopically, however, because at this stage they produce relatively few symptoms which would call attention to their presence.

Biopsy. The important function of the bronchoscope in establishing the diagnosis of bronchogenic carcinoma is to expose the tumor sufficiently to permit removal of tissue by forceps or aspiration for biopsy. In approximately 80 per cent of cases sufficient tissue can be removed with forceps to permit satisfactory study and classification of the tumor. Repeated procedures may be necessary if the lesion is covered with exudate or granulation tissue, or if the tumor has produced extensive pulmonary suppuration which must first be cleared to permit access to the cancerous tissue. Several types of forceps should be available at the time of the bronchoscopy if tissue is to be removed from any of the numerous types of tumors described. Tissue can easily be teased off the verrucous, friable masses which constitute the largest group of tumors with forceps originally designed for the removal of ball-bearings or beads from the air or food passages.[27] These forceps are preferred to the sharper cutting type because they cause little trauma to the tumor bed or to the specimen itself. They are extremely light in weight, allow maximum vision through the bronchoscope during the actual tissue removal, and are delicate enough to permit careful palpation and tactile evaluation of the tissue being removed.

The various types of cup forceps are second in importance in usefulness. The bent-cup forceps permit a side bite which is necessary in the firmer types of tumors invading the bronchial walls. While they are not truly cutting forceps, their heavier construction is of definite value in this type of tissue work. Modifications of this forceps include a sliding blade, and fixed upper blade with a movable up-swinging lower blade. The cutting or punch biopsy types of forceps are standard in almost every set of bronchoscopic equipment. However, their sharp edges and square corners have a tendency to produce unnecessary trauma. Because of this, a rounded cutting biopsy forceps is suggested to overcome these disadvantages.

Considerable care must be exercised in removing tissue with bronchoscopic forceps. The proximity of the great vessels to the bronchi is an anatomical fact. This is even more dramatically apparent when the thoracic viscera are exposed surgically during a pneumonectomy. The destruction of tissue associated with carcinomatous invasion makes perforation of these great vessels a definite possibility,[28] which would be followed by an immediate fatal hemorrhage. Care likewise must be exercised in handling the tissue removed with the biopsy forceps. Such tissues are of necessity very small, usually measuring only a few millimeters in size. They should be placed in a fixing solution immediately. The least crushing in removing them from the forceps or in handling them in solutions used for fixing results in a disarrangement of cell relations which makes histologic study inconclusive. The histology of types of tumors obtained in the 151 positive biopsies is given in Table 2.

TABLE 2.—TYPES OF TUMORS IN 151 POSITIVE BIOPSIES

	No.	%
Squamous cell carcinoma	97	64%
Small round cell carcinoma	25	17%
Anaplastic carcinoma	19	13%
Adeno-carcinoma	10	6%

Special endoscopic problems arise from tumors in the upper lobe bronchi. Should an atelectasis of an entire upper lobe occur, the tumor in many instances protrudes from the upper lobe orifice, and tissue may be obtained without difficulty. However, as the upper lobe bronchus leaves the main bronchus on the right side at an angle of 90 degrees and on the left side at about 70 degrees, a tumor 1 cm. within the orifice can seldom be inspected directly. In many instances the bronchial walls are made even more rigid by the neoplastic involvement and will not permit gentle manipulation with the lip of the bronchoscope to give adequate exposure. These angles, furthermore, are made more acute if the lobe beyond the point of obstruction is atelectatic, thereby tending to pull the bronchus further toward the apex. Thus other aids, such as an artificially induced pneumothorax, are needed to release the distorted bronchus and bring it more parallel to the direction of the main bronchus, facilitating its inspection with the bronchoscope. This procedure was used by Ar-

buckle to obtain the specimen for biopsy in what later proved to be the first case of successful pneumonectomy for bronchogenic carcinoma, the operation being performed by Graham in 1933.[29]

Inspection of the upper lobe bronchi is facilitated further by the use of a retrograde telescope or retrograde mirror. The use of these instruments is hampered by the condensation of moisture on the glass surfaces. By heating them thoroughly first or coating the surfaces with special oils, very satisfactory observations may be made.

Fluoroscopy as an adjunct to the bronchoscopic procedure is routinely used in dealing with certain types of bronchopulmonary foreign bodies. This is especially true in dealing with foreign bodies in the periphery of the lung where the bronchi become so small that the use of bronchoscopes with lumina large enough for direct vision is impossible. Biplane fluoroscopy is then indispensable. Too, it may aid in guiding forceps into the costophrenic angles or upper lobe bronchi in search of tumor tissue for biopsy when the tumor lies in a branch bronchus beyond the scope of direct vision.

Failure to obtain a positive biopsy in cases later proved, either surgically or postmortem, to be bronchogenic carcinoma may be attributed to numerous factors. Most often, the failure is due to the peripheral location of the tumor, or to the fact that it lies in an upper lobe in a position inaccessible to the biopsy forceps. Rigidity of the bronchial tree or the trachea due to the tumor prevents satisfactory exposure of the area involved in other cases. This deformity and rigidity are in themselves of importance in aiding in the establishment of the clinical diagnosis of a malignancy if the tissue is unobtainable, and add to the information gained from the physical and x-ray examinations. Occasionally bronchial compression produced by a large bulky tumor in the parenchyma prevents advance of the bronchoscope to the intrabronchial portion of the tumor. The more advanced lesion is accompanied by copious quantities of sanguinopurulent secretion. At this stage the destruction of tissue confuses the picture by profuse bleeding, pus and granulation tissue. Repeated bronchoscopies may be necessary in these cases to get tissue representative of the tumor itself and not alone of the peripheral inflammatory zone.

Bronchoscopic Criterion of Operability and Inoperability. Besides actually inspecting the endobronchial tumor and noting its character, its exact relationship to surrounding structures, and its extent along a bronchial wall, certain features in the bronchoscopic

appearance of the remainder of the tracheobronchial tree are of great importance. Direct or indirect inspection of the larynx frequently gives valuable correlative information. Hoarseness, produced by a paralysis of the left recurrent laryngeal nerve as it passes under the arch of the aorta, is almost always present in late, left-sided bronchogenic carcinoma. This finding was observed in 21 patients of our series. It is not an infrequent early sign and has occasionally been the first symptom of which the patient complains. Paralysis of the right vocal cord, a much less frequent finding, indicates, in cases of apical tumors on the right side, that invasion or compression has progressed to a point under the right subclavian artery. This again corroborates evidence of extension. Enlargement of the mediastinal glands is detected by a thickening at the division of the trachea. This area, the carina, is usually sharp, regular, and freely movable. With mediastinal metastases, the carina becomes thickened, irregular, and fixed. Should these findings be present, lobectomy or pneumonectomy, of course, is contraindicated.

DIAGNOSTIC PROCEDURES

Table 3 indicates the methods used in obtaining a correct diagnosis in this series of 175 cases. A positive bronchoscopic biopsy was obtained in 136 instances or in 78 per cent of cases. In a few instances a second or even third diagnostic operation was performed. In 39 negative biopsy cases, a definite compression stenosis of the bronchi, distortion of the carina, or alteration in the appearance of the mucous membrane of the lower air passage was noted in 11 cases; the findings were considered typical enough of bronchogenic carcinoma to confirm the diagnosis. Exploratory thoracotomy established a diagnosis in 5. Three of these had a pneumonectomy and two were found to be inoperable due to an extensive dissemination. Pleural fluids and metastatic glands yielded carcinomatous cells in 3 cases each; sputum, coughed out tissue and aspiration biopsy[30] were positive in one case each. Evidences from x-ray and clinical data were deemed sufficient to justify a diagnosis in 14 in whom bronchoscopy was negative or for some reason was contraindicated.

TREATMENT

The treatment of bronchogenic carcinoma is primarily surgical. The tremendous strides made during the past ten years in this surgical management are evident from a review of the literature.[4, 31-33] In every case in this series considered operable from a clinical and bronchoscopic viewpoint, surgery was recommended. However, the pa-

tients comprising the series were referred principally for bronchoscopic examination and diagnosis. Consequently, an accurate record of the number operated on cannot be obtained, since the operations, either exploratory or therapeutic, were done elsewhere in many instances. Nevertheless, as Clerf[34] states, a remarkable disproportion exists between the large number of reported cases of primary carcinoma of the bronchus and the small group that has been successfully treated.

TABLE 3.—METHOD OF ESTABLISHING DIAGNOSIS IN 175 CASES OF PRIMARY BRONCHOGENIC CARCINOMA

	No.	%
Positive Bronchoscopic Biopsy	136	78%
Characteristic Alteration of Trachea or Bronchi	11	6%
Exploratory Thoracotomy	5	3%
Pleural Fluid Aspiration	3	2%
Biopsy of Metastatic Nodes	3	2%
Sputum Biopsy	2	1%
Needle Aspiration of Tumor	1	
X-ray and Clinical Diagnosis	14	8%

Bronchoscopic treatment of bronchogenic carcinoma is mentioned here not as opposed to surgery but as a means of therapy in patients otherwise inoperable. This interesting phase of bronchology is directed toward the reestablishment of the bronchial lumen and efforts to keep it patent. Forceps removal of a benign bronchial tumor was described first by von Eicken[35] in 1907. Later Jackson[36] removed a tumor from the right main bronchus by the same method. The tumor removed by von Eicken was considered benign; that removed by Jackson was regarded, pathologically, as malignant. The forceps removal of tissue obstructing a bronchus is a practical, rapid method of reestablishing the lumen. The control of hemorrhage is difficult with this technique, however; consequently, it has been supplanted to a large degree by the use of surgical diathermy. Vinson and Bowing[37] first described the value of this type of therapy which, in certain selected cases, is of definite value in the treatment of primary carcinoma of the bronchus.[38-40]

A more recent type of endobronchial therapy directed bronchoscopically is the implantation of radon seeds or radium directly into

the obstructing tumor. Although the results have been palliative in most instances, a few exceptions should be mentioned. Negus,[41] Kernan,[39] Ormerod,[42] and others have reported clinical cures by the use of this method together with the use of surgical diathermy. It should be noted that in most of these cases the biopsy revealed the tumor to be an adenocarcinoma.

The bronchoscopic insertion of tubes containing radon has been suggested by Soulas,[43] von Eicken,[44] Negus,[41] and others. More recently, Pressman and Emery[45] have devised an applicator for the implantation of radium needles directly into the bronchus to remain in place 30 to 65 hours. A preliminary pneumothorax was of definite advantage in this latter type of therapy.

Clinical cures of bronchogenic carcinoma through the use of any or all of these bronchoscopically directed methods of therapy are significant and frequent enough to suggest their use in selected cases otherwise inoperable.

Roentgen therapy has likewise received much consideration. In our experience it has been of assistance in alleviating bronchial obstruction in some instances and thus giving palliative relief to the patient. Hammond[46] and Craver,[47] however, have reported more significant results in isolated instances.

These isolated instances of favorable results by either bronchoscopic cautery or irradiation should not be considered as an alternative to the surgical removal of the lung in any case considered operable.

700 North Michigan Avenue.
Hinsdale Sanatorium, Hinsdale, Ill.
1439 South Michigan Avenue.

REFERENCES

1. Deaths and Death Rates for Cancer and Other Malignant Tumors, United States: 1940, Bureau of the Census Vital Statistics, Special Reports 15:34-391 (June 16) 1942.

2. Matz, P. B.: The Incidence of Primary Bronchiogenic Carcinoma, J. A. M. A. 111: 2086 (Dec. 3) 1938.

3. Brines, O. A., and Kenning, J. C.: Bronchiogenic Carcinoma, Am. J. Clin. Path. 7:120 (Mar.) 1937.

4. Ochsner, Alton, and DeBakey, Michael: Surgical Considerations of Primary Carcinoma of the Lung, Surgery 8:992 (Dec.) 1940.

5. Simons, E. J.: Primary Carcinoma of the Lungs, Chicago, Year Book Publishers, 1937.

6. Loizaga, N. S.: Del Carcinoma Primitivo Broncopulmonar, Libreria y Editorial "El Ateneo", Buenos Aires, 1938.

7. Fine, M. J., and Jaso, J. V.: Silicosis and Primary Carcinoma of the Bronchus, J. A. M. A. 104:40 (Jan. 5) 1935.

8. Mulligan, R. M., and Harper, F. R.: The Morphology of Primary Carcinoma of the Human Lung, J. Thoracic Surg. 12:734 (Dec.) 1943.

9. Vorwald, A. J., and Karr, J. W.: Pneumonoconiosis and Pulmonary Cancer, Am. J. Path. 14:49, 1938.

10. Arkin, Aaron, and Wagner, D. H.: Primary Carcinoma of the Lung, J. A. M. A. 106:587 (Feb. 22) 1936.

11. Wells, H. G.: Cancer Statistics as They Appear to a Pathologist, J. A. M. A. 88:476 (Feb. 12) 1927.

12. Stephens, H. B.: Primary Carcinoma of the Lung, Amer. J. Surg. 56:201 (April) 1942.

13. Singer, J. J.: Primary Bronchiogenic Carcinoma, Surgery 8:910 (Dec.) 1940.

14. Perrone, J. A., and Levison, J. P.: Primary Carcinoma of the Lung (Report of 115 Cases, 38 Autopsies and 77 Bronchoscopic Biopsies), Ann. Int. Med. 17:12 (July) 1942.

15. Holman, Emil: Carcinoma of the Lung Simulating Inflammatory Disease, J. A. M. A. 113:108 (July) 1939.

16. Halpert, Bela: Morphologic Aspects of Carcinoma of the Lung, Surgery 8:903 (Dec.) 1940.

17. Quinland, W. S.: Bronchiogenic Carcinoma, South. M. J. 35:729 (Aug.) 1943.

18. Kikuth, W.: Uber Lungecarcinom., Virchows Arch. f. path. Anat. 255: 107, 1925.

19. Holinger, P. H., and Rigby, Ralph: Bronchogenic Carcinoma Without Bronchial Obstruction, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 50:1270 (Dec.) 1941.

20. Jackson, Chevalier, and Jackson, C. L.: Bronchoscopy, Esophagoscopy and Gastroscopy, Philadelphia, W. B. Saunders Co., 1934.

21. Hartung, A., and Wachowski, T. J.: Diagnosis of Bronchogenic Carcinoma, Illinois M. J., Nov., 1937.

22. Jenkinson, E. L., and Hunter, A. F.: Bronchiogenic Carcinoma, J. A. M. A. 113:2392 (Dec. 30) 1937.

23. Olds, J. W., and Kirklin, B. R.: Primary Carcinoma of the Lung: Roentgenologic Study of 206 Proved Cases, Am. J. Roentgenol. & Rad. Therapy, 19:357 (Sept.) 1940.

24. Farinas, P. L.: Malignant Bronchial Stenosis: Bronchographic Aspect, Am. J. Roentgenol. & Rad. Therapy 15:180 (Aug.) 1938.

25. Holinger, Paul, and Andrews, A. H., Jr.: Bronchial Obstruction, Am. J. Surg. 54:193 (Oct.) 1941.

26. Holinger, Paul, and Radner, D. B.: Bronchoscopic Diagnosis of Bronchial Carcinoma, Surgery 8:939 (Dec.) 1940.

27. Jackson, C. L., and Konzelmann, F. W.: Bronchial Carcinoma, J. Thoracic Surg. 4:165 (Dec.) 1934.

28. Lederer, F. L.: Hemorrhage in Lung Cancer: A Fatal Case Following Bronchoscopy, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 44:157 (Mar.) 1935.

29. Graham, E. A., and Singer, J. J.: Successful Removal of an Entire Lung for Carcinoma of Bronchus, J. A. M. A. 101:1371, 1933.

30. Martin, H. E., and Ellis, E. B.: Aspiration Biopsy, Surg., Gynec. & Obst. 59:578 (Oct.) 1934.

31. Overholt, R. H., and Rumel, W. R.: Clinical Studies of Primary Carcinoma of the Lung, J. A. M. A. 114:735 (Mar. 2) 1940.

32. Harrington, S. W.: Pneumonectomy for Carcinoma of the Lung, J. Thoracic Surg. 11:396 (April) 1942.

33. Shenstone, N. S.: Experiences with Total Pneumonectomy, J. Thoracic Surg. 11:405 (April) 1942.

34. Clerf, L. H.: Carcinoma of the Bronchus, Radiology 28:438, 1937.

35. Von Eicken, C.: Bronchoscopische Mitteilungen, Verhandl. d. Ver. suddeutsch., Laryngol. p. 410, 1907.

36. Jackson, Chevalier: Endothelioma of the Right Bronchus Removed by Peroral Bronchoscopy, Am. J. M. Sc. 152:371, 1917.

37. Vinson, P. P., and Bowing, H. H.: Surgical Diathermy for Tumors of the Trachea, Laryngoscope 36:217, 1926.

38. Jackson, C. L.: The Bronchoscopic Diagnosis and Treatment of Bronchial Tumors, Ztschr. f. Hals-, Nasen- u. Ohrenh. 40:622, 1937.

39. Kernan, J. D.: Treatment of a Series of Cases of So-Called Carcinoid Tumors of the Bronchi by Diathermy: A Report of Ten Cases, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 44:1167, 1935.

40. Moersch, H. J., and Bowing, H. H.: Primary Carcinoma of Bronchus Treated Successfully with Surgical Diathermy, Ann. Surg. 102:989, 1935.

41. Negus, V. E.: Intrathoracic New Growths and the Value of Bronchoscopy in Diagnosis and Treatment, J. Laryng. & Otol. 48:457, 1933.

42. Ormerod, F. C.: Malignant Disease of the Bronchus, J. Laryng. & Otol. 48:733, 1933.

43. Soulas, Andre: Le diagnostic bronchoscopique des cancers broncho-pulmonaires, Bull. et mém. Soc. méd. d. hôp. de Paris 47:1536, 1931.

44. Von Eicken, Carl: Bronchoskopische Behandlung eines Bronchuskarzinoms, Sitzungsb. d. Oto-laryngol. Gesellsch. z. Berlin, p. 71, 1933.

45. Pressman, J. J., and Emery, C. K.: A New Method of Radium Application in Cancer of the Bronchus, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 46:314, 1937.

46. Hammond, A. E.: Diagnosis and Treatment of Carcinoma of the Lung, Arch. Otolaryng. 31:780 (May) 1940.

47. Craver, L. F.: Bronchiogenic Carcinoma: Am. J. Roentgenol. & Rad. Therapy 43:469 (April) 1940.

II

MALIGNANT EXOPHTHALMOS

LeRoy A. Schall, M.D.

AND

Daniel J. Reagan, M.D.

Boston, Mass.

It is well known that a certain type of Graves' disease in which the eye signs predominate has, in some cases, necessitated surgical orbital decompression for the relief of symptoms and the preservation of vision. This type of case, variously referred to as "malignant exophthalmos," "exophthalmic ophthalmoplegia," "progressive postoperative exophthalmos with low metabolic rate,"[5] usually follows thyroidectomy but occasionally occurs primarily. Means[5] and his colleagues have adopted the practice of dividing Graves' disease into the classic type and the special ophthalmopathic type with which this paper is principally concerned.

The etiological concept, generally accepted, of the ophthalmic picture in the malignant type of exophthalmos is based partly on fact and partly on theory.[5] It is known that in the classic type of Graves' disease there is an increased activity on the part of the thyroid which produces an excess of its hormone and the clinical picture of hyperthyroidism. It is also known that the thyrotropic hormone of the anterior pituitary is manufactured in excess but is in turn inhibited by the large amounts of thyroxin which its stimulation of the thyroid has produced. Thus, "the pituitary-thyroid axis," so-called, is reared to an abnormally high level of activity. It is theorized that some stimulus from the hypothalamus initiates the process in the anterior pituitary. Many workers have demonstrated experimentally that injections of large amounts of the thyrotropic principle of the anterior pituitary will produce Graves' disease in animals and that thyroidec-

---

From the Department of Otolaryngology of the Massachusetts Eye and Ear Infirmary, Boston, Mass.

Read before the Eastern Section of the American Laryngological, Rhinological and Otological Society, Philadelphia, Pa., Jan. 12, 1945.

tomy, in conjunction with a continuation of these injections, will very often produce the picture of malignant exophthalmos or the ophthalmopathic type of Graves' disease. Therefore, to theorize, malignant exophthalmos is probably due to a lack of thyroid response to the excess amounts of TSH, or a removal of the thyroid inhibition of TSH by thyroidectomy. That there are other factors concerned is obvious, because few people undergoing thyroidectomy for Graves' disease develop malignant exophthalmos. Whether this added factor is concerned with gonads, as Marine[4] has postulated, or whether the anterior pituitary fails to revert to a normal level of activity, as it would seem to do in the ordinary case, is problematical.

The pathology in the orbit, described in detail initially by Naffziger in 1932[7] and since elaborated upon by other workers, particularly by the many researches of Smelser, is composed of generalized edema and lymphocytic infiltration of the orbital contents, especially the extra-ocular muscles which are many times their normal size. The muscles undergo a Zenker's type of degeneration and may be replaced to some degree by fibrous tissue. Galli-Mainini[2] suggests that the eyeball is situated in the orbit much as a cork in a bottle and that the muscle changes, weakening their power, in company with an increased orbital content, result in exophthalmos.

The clinical picture of malignant exophthalmos is striking. The patient may complain of burning, lacrimation, photophobia, deep retrobulbar pain, and a sense of pressure within the orbits. Some visual loss often occurs, apparently due to changes around the optic nerve with resultant pressure and tension on it. Mulvany[6] has found that the nerve itself is normal. Papilledema and peripapillary hemorrhages may be observed on fundus examination. Excursions of the eyeball are usually limited in all fields of gaze especially upward, and troublesome diplopia often ensues. In the advanced case there are edema of the lids and conjunctiva, hyperemia of the latter, and, on pressure, the globe imparts to the fingers a sense of firm retrobulbar resistance unlike that felt on pressing against the normal eyeball. In the most advanced cases, the patient is unable to close the lids, keratitis and ulceration follow, and endophthalmitis may threaten both loss of vision and life. These malignant types are usually found in patients who have had a thyroidectomy. Occasionally there is an idiopathic case. The chief differences between this ophthalmopathic type and the classic Graves' disease are as follows:

	OPHTHALMOPATHIC	CLASSIC
1. Sex Incidence	50-50	Female 9-1
2. Hyperthyroid Symptoms	Little or none. May be hypothyroid.	Moderate or marked
3. Effect of Iodine and Operation (Thyroidectomy)	Does no good. May make them worse.	Usually markedly improved
4. Exophthalmos	Average 7 mm.	Average 2.5 mm.
5. Basal Metabolic Rate	Normal, slightly elevated, or low	High

Treatment of this condition has been of three types. Occasional cases have responded to or been held in abeyance by the administration of thyroid extract or x-ray treatment directed to the orbits. If these methods fail, surgical decompression of the orbit according to Naffziger's technique[7] has been quite successful. Briefly, this operation consists of creating two frontal bone flaps by means of which the orbital roofs are exposed and removed as far back as the optic foramen. The latter may also be decompressed if the swelling around the nerve at the time of operation would seem to warrant it. The anterior rim of the orbit is preserved to maintain the contour of the brow, and none of the nasal sinuses are entered. By this method, a potential area of expansion for the orbital contents is provided; and, over several months' time, this takes place into the anterior cranial fossa, usually with satisfactory regression of the clinical signs and symptoms.

In 1936 Sewall[9] suggested, following anatomical studies, that the nasal sinuses could be utilized for orbital decompression and thus provide an actual rather than a potential space. He recommended the removal of the ethmoid sinus, the floor of the frontal, and in some cases, the medial floor of the orbit as far laterally as the infra-orbital vessels. In 1938 Kistner[3] reported two patients treated in this manner with satisfactory results. This is the only report we could find in the literature concerning this type of orbital decompression. Both of Kistner's patients developed progressive exophthalmos several months after thyroidectomy. The first patient had pain, diplopia, and partial bilateral visual loss. By removing the frontal sinus floor and the ethmoid sinus, particularly the lamina papyracea as far back as the optic foramen and also incising the orbital fascia, Kistner was able to effect a recession of the exophthalmos of 4-5 mm. in three weeks, complete subsidence of symptoms in a few days, and a grad-

ual improvement in vision. The second patient had developed a corneal ulcer as well as the classic symptoms mentioned above. Four weeks following the same type of operation the proptosis had returned to normal, the symptoms had disappeared, the ulcer was healed, and there was a continuing improvement in vision. Kistner lists the advantages of this type of decompression as follows:

1. It is less disfiguring and formidable than the Naffziger operation.

2. An actual rather than a potential space is created.

3. Sometimes the roof of the orbit is occupied by extension of air cells which the Naffziger technique avoids entering.

4. There is no subjective and objective evidence of pulsation in the orbital contents which may follow the intracranial operation.

We would like to report two cases of malignant exophthalmos treated by bilateral external ethmoidectomies with removal of the lamina papyracea as far back as the sphenoid sinus.

REPORT OF CASES

CASE 1. Male, age 55, referred to one of us (L.A.S.) by Dr. James M. Murphy of Palmer, Mass., for polypoid ethmoiditis and exophthalmos of unknown etiology. He had recently returned from Iceland and the patient thought that the glare of the sun had affected his eyes. It was apparent that the exophthalmos was the major problem and he was admitted to the Massachusetts General Hospital under the care of Dr. James H. Means. Studies revealed a picture consistent with malignant exophthalmos, basal metabolic rates of +3 and +15, and questionable symptoms of borderline hyperthyroidism. The patient had never undergone thyroid surgery. A course of x-ray therapy to the orbits, concluded 18 days before transfer back to the Massachusetts Eye and Ear Infirmary on April 5, 1944, had produced no subjective or objective change in the exophthalmos.

Exophthalmometer readings at the time of admission to the Infirmary were: O.D. 27.; O.S. 25.5. The patient complained of tearing, burning, deep retrobulbar pain, with a sense of pressure and intermittent blurring of vision. The eyes showed edema of the lids and conjunctiva with hyperemia of the latter, exophthalmos, and some limitation of global motion in all fields of gaze. The cornea and optic discs were normal. There was a marked sense of resistance on inward pressure against the eyeballs.

Clinically and radiologically the patient showed evidence of bilateral polypoid ethmoiditis and three days after admission, following

the institution of sulfadiazine therapy, bilateral external ethmoidectomies were performed by Drs. D. J. Reagan and D. K. Lewis. In an attempt to provide some degree of orbital decompression, the lateral ethmoid walls were removed as far back as the sphenoid sinuses. The postoperative convalescence was satisfactory for ten days, during which time the patient's ocular as well as nasal symptoms disappeared, and there was a 2 mm. recession of the eyes. Thirteen days after operation and three days following the removal of sulfadiazine therapy the patient developed a pneumococcus meningitis, coincident with a super-imposed rhinitis. This responded completely to the reestablishment of sulfadiazine treatment and was well within a week. At discharge, the patient had no symptoms referable to his exophthalmos, and the eyes had receded into the orbits 3 mm. each. Objectively, there was some persistence of the edema and thickening of the lids and slight edema of the bulbar conjunctiva O.D. externally.

CASE 2. A 45-year-old woman entered the Massachusetts General Hospital, under the care of Dr. James H. Means, in May 1944, with the history of progressive bilateral exophthalmos, lacrimation, partial loss of global motion, and diminished visual acuity of five months' duration. She had mild symptoms of hyperthyroidism, with basal metabolic rates of +29 and +32. Her thyroid was two to three times the normal size, and she had never had a thyroid operation.

Ocular examination revealed edema of the lids and conjunctiva, limitation of motion in all fields of gaze, exophthalmos, and a firm sense of retrobulbar resistance when pressure was applied to the globes. Exophthalmometer readings were O.D. 28 mm., O.S. 25 mm. Both optic discs were blurred, with peripapillary hemorrhages in the right disc.

During the succeeding several days she developed bilateral exposure keratitis and, although being treated with iodine and x-ray directed to the orbits, it was decided to do bilateral ethmoidectomies because of the success of our first case. The sinuses were normal by x-ray examination. The right cornea became ulcerated and the lids were sutured by Dr. David Cogan to protect the cornea. External ethmoidectomies were done under local anesthesia (L.A.S.) with removal of the os planum as far back as the sphenoid. The ethmoidal labyrinths were exenterated from the orbital side but the medial nasal wall of the middle meatus was not removed. The middle turbinate was left in place, so that the physiology of the nose was not disturbed.

Eight days postoperatively, marked subjective improvement was noted for the first time and it was found that the exophthalmos had

receded 2 mm. At discharge, approximately three weeks following surgery, the recession, O.U., was noted to be 3 mm. and the patient was symptom free. Some edema and thickening of the lids were present and the patient still presented to some degree an exophthalmic appearance.

CONCLUSIONS

Two cases of malignant exophthalmos are presented that were treated by decompression into the sinuses following the method proposed by Sewall[9] and first used by Kistner.[3] We agree with Kistner's conclusions that this operation is:

1. Less disfiguring and formidable than the Naffziger operation.

2. An actual rather than a potential space is created.

3. Sometimes the roof of the orbit is occupied by extension of air cells which the Naffziger technique avoids entering.

4. There is no subjective and objective evidence of pulsation in the orbital contents which may follow the intracranial operation.

243 CHARLES STREET.

REFERENCES

1. Cogan, D. G.: Medical Progress—Ophthalmology, New England J. Med. 222:716-720, 1940.

2. Galli-Mainini, C.: Exophthalmometric Measurements In Patients With Thyroid Disease With Some Discussion of Their Significance, Ann. Int. Med. 16:415, 1942.

3. Kistner, F. B.: Decompression for Exophthalmos, J. A. M. A. 112:37, 1939.

4. Marine, D.: Studies of the Pathological Physiology of the Exophthalmos of Graves' Disease, Ann. Int. Med. 12:443, 1938.

5. Means, J. H.: The Nature of Graves' Disease with Special Reference to its Ophthalmic Component, Am. J. M. Sc. 207:1-19 (Jan.) 1944.

6. Mulvany, J. H.: Exophthalmos of Hyperthyroidism, Am. J. Ophth. 27:589 (June) 1944.

7. Naffziger, H. C., and Jones, O. W.: The Surgical Treatment of Progressive Exophthalmos Following Thyroidectomy, J. A. M. A. 99:638, 1932.

8. Naffziger, H. C.: Progressive Exophthalmos After Thyroidectomy, West. J. Surg. 40:530 (Oct.) 1932.

9. Sewall, E. C.: Operative Control of Progressive Exophthalmos, Arch. Otolaryng. 24:621 (Nov.) 1936.

III

NASAL ALLERGY FOR THE PRACTICING RHINOLOGIST

GEORGE E. SHAMBAUGH, JR., M.D.

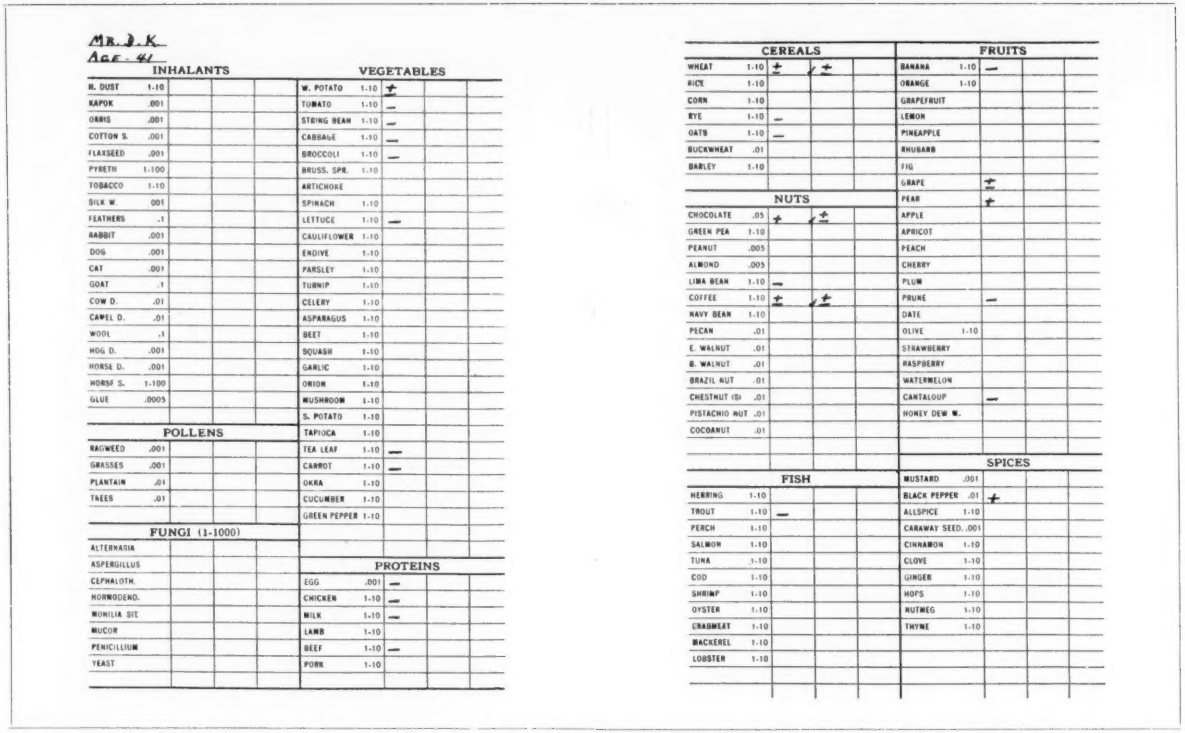
CHICAGO, ILL.

I. PATHOLOGIC AND THEORETIC CONSIDERATIONS UNDERLYING NASAL ALLERGY

The remarkable ability of the nasal and sinus mucosa to recover spontaneously and completely from severe infections may be observed in the severe acute purulent streptococcic rhinitis and sinusitis that frequently complicate scarlet fever. In sharp contrast to this normal tendency to spontaneous recovery is the inability of the nasal and sinus mucosa in other persons to throw off and recover from the very mildest type of low-grade infection, particularly when hyperplastic and polypoid changes are present in the ethmoid and other sinus regions. It has been only recently that we have begun to learn that there is an underlying allergy in these cases.

At least 70 percent of chronic sinus infections, and at least 90 per cent of chronic nasal infections can be shown to have an underlying allergic factor responsible for the chronicity. The therapeutic results that can be achieved in chronic nasal and sinus diseases by combining the allergic treatment with the treatment of the underlying infection far surpass the results that can be obtained when either factor is neglected. Since it is impractical for the average patient to make repeated visits to two different physicians for his nasal or sinus disease, the best therapeutic results will be obtained, we believe, when the rhinologist makes his own allergic studies in these cases.

The importance of the allergic factor in chronic nasal and sinus infection has only recently begun to be recognized. To Hansel belongs the chief credit for directing the attention of the rhinologist to the frequent allergic factor. Allergy, itself, is so new a specialty that the word itself was unknown until 1906, when von Pirquet used it to describe this curiously altered capacity to react to those things with which we come into contact. Translated from the Greek, it means literally "altered energy" or "altered reactivity".



Mr. J. K.
Age - 41

INHALANTS

H. DUST	1-10				
KAPOK	.001				
ORRIS	.001				
COTTON S.	.001				
FLAXSEED	.001				
PYRETH	1-100				
TOBACCO	1-10				
SILK W.	.001				
FEATHERS	.1				
RABBIT	.001				
DOG	.001				
CAT	.001				
GOAT	.1				
COW D.	.01				
CAMEL D.	.01				
WOOL	.1				
HOG D.	.001				
HORSE D.	.001				
HORSE S.	1-100				
GLUE	.0005				

POLLENS

RAGWEED	.001				
GRASSES	.001				
PLANTAIN	.01				
TREES	.01				

FUNGI (1-1000)

ALTERNARIA					
ASPERGILLUS					
CEPHALOTH.					
HORMODEND.					
MONILIA SIT.					
MUCOR					
PENICILLIUM					
YEAST					

VEGETABLES

W. POTATO	1-10	+			
TOMATO	1-10	−			
STRING BEAN	1-10	−			
CABBAGE	1-10	−			
BROCCOLI	1-10	−			
BRUSS. SPR.	1-10				
ARTICHOKE					
SPINACH	1-10				
LETTUCE	1-10	−			
CAULIFLOWER	1-10				
ENDIVE	1-10				
PARSLEY	1-10				
TURNIP	1-10				
CELERY	1-10				
ASPARAGUS	1-10				
BEET	1-10				
SQUASH	1-10				
GARLIC	1-10				
ONION	1-10				
MUSHROOM	1-10				
S. POTATO	1-10				
TAPIOCA	1-10				
TEA LEAF	1-10	−			
CARROT	1-10	−			
OKRA	1-10				
CUCUMBER	1-10				
GREEN PEPPER	1-10				

PROTEINS

EGG	.001	−			
CHICKEN	1-10	−			
MILK	1-10	−			
LAMB	1-10				
BEEF	1-10	−			
PORK	1-10				

CEREALS

WHEAT	1-10	+	+		
RICE	1-10				
CORN	1-10				
RYE	1-10	−			
OATS	1-10	−			
BUCKWHEAT	.01				
BARLEY	1-10				

NUTS

CHOCOLATE	.05	+	+		
GREEN PEA	1-10				
PEANUT	.005				
ALMOND	.005				
LIMA BEAN	1-10	−			
COFFEE	1-10	+	+		
NAVY BEAN	1-10				
PECAN	.01				
E. WALNUT	.01				
B. WALNUT	.01				
BRAZIL NUT	.01				
CHESTNUT (B	.01				
PISTACHIO NUT	.01				
COCOANUT	.01				

FISH

HERRING	1-10				
TROUT	1-10	−			
PERCH	1-10				
SALMON	1-10				
TUNA	1-10				
COD	1-10				
SHRIMP	1-10				
OYSTER	1-10				
CRABMEAT	1-10				
MACKEREL	1-10				
LOBSTER	1-10				

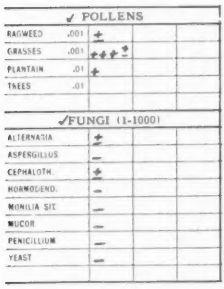
FRUITS

BANANA	1-10	−			
ORANGE	1-10				
GRAPEFRUIT					
LEMON					
PINEAPPLE					
RHUBARB					
FIG					
GRAPE		+			
PEAR		+			
APPLE					
APRICOT					
PEACH					
CHERRY					
PLUM					
PRUNE		−			
DATE					
OLIVE	1-10				
STRAWBERRY					
RASPBERRY					
WATERMELON					
CANTALOUP		−			
HONEY DEW M.					

SPICES

MUSTARD	.001				
BLACK PEPPER	.01	+			
ALLSPICE	1-10				
CARAWAY SEED	.001				
CINNAMON	1-10				
CLOVE	1-10				
GINGER	1-10				
HOPS	1-10				
NUTMEG	1-10				
THYME	1-10				

Fig. 1.—Case of Allergy to Coffee - Headaches.



✓ POLLENS				
RAGWEED	.001	+		
GRASSES	.001	++++		
PLANTAIN	.01	+		
TREES	.01			
✓FUNGI (1-1000)				
ALTERNARIA		+		
ASPERGILLUS		–		
CEPHALOTH.		+		
HORMODEND.		–		
MONILIA SIT.		–		
MUCOR		–		
PENICILLIUM		–		
YEAST		–		

Fig. 2.—Case of Grass Pollen Allergy, Mr. E. J. M., age 32.

An example of allergy to foods is shown in the case of Mr. D. K., age 41, who complained of frequent headaches for 10 years, continuous for four months. Intradermal tests (Fig. 1) with common foods showed slight positive reactions to wheat, chocolate and coffee. On omitting these foods, headaches disappeared, with no recurrence on adding wheat and chocolate, but headaches recurred when coffee was added. The patient now remembers coffee has always caused "heart burn." Since omitting coffee from his diet, headaches have been relieved. As coffee in the normal individual does not cause headache or "heart burn," this is an instance of "altered reactivity", or allergy.

The allergic reaction is believed to be an immune reaction, the result of the union of specific antibodies with the particular substance to which the person is allergic. The specific antibodies are produced in the body after previous exposure to the particular substance known as the antigen or allergen. Most allergens are protein, but some are non-protein, as in the case of drug allergies. The specific antibody causing the allergic reaction is found in the blood serum and the tissues throughout the body, but particularly in the skin and the mucous membranes. These are the tissues which are in contact with the outer world, and in which immune reactions are most highly developed as a protective mechanism. The tissues in which the allergic reaction occurs, and from which the allergic symptoms arise, are called shock tissues. In most pollen allergies the specific antibody can be demonstrated in the skin by introducing the pollen intradermally. This antibody is called a reagin, and such persons are said to have a reaginic

INHALANTS

H. DUST	1-10	—			
KAPOK	.001	—			
ORRIS	.001	±			
COTTON S.	.001	—			
FLAXSEED	.001	—			
PYRETH	1-100	—			
TOBACCO	1-10	±			
SILK W.	.001	—			
FEATHERS	.1	—			
RABBIT	.001	—			
DOG	.001	—			
CAT	.001	—			
GOAT	.1	—			
COW D.	.01	±			
CAMEL D.	.01	—			
WOOL	.1	±			
HOG D.	.001	—			
HORSE D.	.001				
HORSE S.	1-100				
GLUE	.0005				

Fig. 3.—Case of Dust Allergy, Mrs. M. S., age 42.

INHALANTS

H. DUST	1-10	++			
KAPOK	.001	—			
ORRIS	.001	—			
COTTON S.	.001	—			
FLAXSEED	.001	—			
PYRETH	1-100	—			
TOBACCO	1-10	—			
SILK W.	.001	—			
FEATHERS	.1	±			
RABBIT	.001	—			
DOG	.001	—			
CAT	.001	—			
GOAT	.1	±			
COW D.	.01	±			
CAMEL D.	.01	±			
WOOL	.1	±			
HOG D.	.001	—			
HORSE D.	.001	—			
HORSE S.	1-100	—			
GLUE	.0005	—			

Fig. 4.—Case of Dust Allergy, Miss B. S., age 44.

allergy. In most cases of food allergy the skin tests are negative, and the individual is said to have a non-reaginic allergy. Nevertheless, though unable to demonstrate the specific antibody by skin tests, we believe that the mechanism of the allergic reaction is essentially the same in reaginic and non-reaginic allergy, namely an antigen-antibody reaction.

An instance of reaginic allergy is shown in the case of Mr. E. J. M., age 32, who complained of sneezing and profuse watery nasal discharge occurring each June. The results of the intradermal tests with the common pollens and fungi are given in Fig. 2.

The following is a case of non-reaginic allergy: Mrs. M. S., age 42, had symptoms of excessive sneezing for several years, worse indoors, and when stirring up dust doing housework (Fig. 3). The intradermal test to house dust was negative, but her symptoms were completely relieved after two injections of house dust extract, 0.05 cc. of 1:100,000 solution, and the patient was discharged.

We do not know what in the allergic individual makes him develop antibodies against substances which normally do not stimulate antibody production and the consequent symptoms, except that heredity is an important factor.

The pathology of the allergic reaction is seen in the positive skin test. The allergic reaction should be regarded as a defense reaction, or immune reaction, against an invader. The union between allergen and antibody in the tissues is the primary immunologic response designed to anchor or fix the allergen, and thus prevent the invader from spreading deeper into the body. The secondary immunologic response is different with different tissues, but appears to be the result of the action of histamine, liberated by the union of allergen and antibody. In the skin there is local inflammation with increased capillary permeability and edema. In mucous membranes there is, in addition, hyperactivity of the mucous glands, and in the intestine and bronchi, smooth muscle spasm is added to the edema and hypersecretion of mucus. The third stage is the destruction of the allergen in the inflamed area by proteolytic substances and phagocytes, chiefly eosinophiles.

Nasal allergy in its early stages is a reversible reaction, so that after the hay fever season, the "waterlogged" nose usually returns entirely to normal. Usually, but not always, for sometimes the hay fever is so severe that permanent ethmoid polypi persist long after the pollen has left the air. Secondary infection is prone to occur in the chronically allergic nose, partly because of mechanically impaired drainage; but the allergic reaction, itself, appears to lower the resistance of the tissues to infection: Miss B. S., age 44, complained of constant soreness in the pharynx, worse during the winter, and worse when exposed to dust. The pharynx was slightly reddened. A culture of the secretions showed hemolytic streptococcus. Intradermal dust reaction was two-plus (Fig. 4). After two injections of 0.05 cc. of 1:1,000,000 house dust extract, the chronic sore throat was completely relieved.

Long-standing nasal allergy, when complicated by infection, results in irreversible hyperplastic and polypoid changes in the nasal and sinus mucosa. In such cases surgical removal of nasal polypi, of mulberry posterior tips of the inferior turbinates, and occasionally removal of a hyperplastic infected sinus mucosa must be combined with allergic management to effect a cure. Experience has shown again and again that the most radical sinus surgery with complete removal of every shred of mucosa from all of the sinuses may fail to effect a permanent cure if the allergic factor is neglected or overlooked. In these cases regeneration of the sinus mucosa is often followed by a recurrence of the allergic reactions and secondary infection of this regenerated mucosa. Moreover, the mucosa of the nasal passages,

TABLE 1.

	PURE INFECTION CHRONIC SINUSITIS	ALLERGIC CHRONIC SINUSITIS
Comparative Frequency	30% (or less)	70% (or less)
Etiology	Anaerobic type of streptococcus	Infection with staph. or strep. secondary to chronic allergic edema with impaired drainage.
Onset	(a) Follows an abscessed upper molar tooth (b) Follows a severe respiratory infection such as scarlet fever	Insidious or may date from an ordinary head cold.
Sinuses Involved	Maxillary most often. Tends to be confined to one side of the nose	Ethmoid sinus most often involved. Tends to be bilateral and to involve all of the sinuses more or less.
Nasal Mucosa	Unchanged except at the sinus ostium where it is inflamed and may be hypertrophied.	Same changes as in the sinuses: edema, polypoid changes, excessive secretion of mucus.
Symptoms	Minimal: scant postnasal discharge. Occasional foul odor, rarely headaches. Systemic symptoms frequent: arthritis, neuritis, etc.	Maximal: nasal blocking, sneezing, coughing. Profuse mucopurulent nasal and postnasal discharge. Headaches frequent and severe. Fatigue, lassitude and sometimes G. I. symptoms.
Histology of Sinus Mucosa	Epithelium normal. Subepithelial connective tissue slightly thickened with infiltration with plasma cells and polymorphonuclear leukocytes	Epithelium desquamating, increased number of goblet-cells, basement membrane thickened. Edema of subepithelial connective tissue. infiltrated with eosinophiles in addition to plasma cells and polymorphonuclear leukocytes.
Irrigation	Liquid pus, often very foul	Gelatinous or tenacious mucopus.
Cytology of Pus	Neutrophiles only	Few to many eosinophiles mixed with the neutrophiles.

TABLE 1.—(*Continued*)

	PURE INFECTION CHRONIC SINUSITIS	ALLERGIC CHRONIC SINUSITIS
Treatment	Irrigations may effect a cure, but *ventilation* usually required by means of a window operation.	Remove the allergic factor and the infection tends to clear up, or responds rapidly to a few irrigations or to surgery.
Prognosis	Excellent. Once cleared does not recur.	Poor if allergic factor is untreated. Good if allergic factor is removed and the infection is treated simultaneously.

trachea and bronchi is always more or less involved in the hyperplastic changes and cannot be extirpated surgically.

Cases of chronic sinusitis can be differentiated into two clear-cut and distinct etiologic types. In one the chronicity is due to an anaerobic type of infecting organism; in the other, the chronicity is due to a chronic allergy with secondary infection. In Table 1 the characteristics which serve to differentiate the two types are contrasted.

SUMMARY

The normal nasal and sinus mucosa is able to recover spontaneously from most acute infections. The allergic sinus mucosa may be unable to throw off even mild acute infections, which then proceed to become chronic. An underlying nasal allergy is responsible for the chronicity of at least 70 per cent of all cases of chronic sinusitis, and at least 90 per cent of all cases of chronic rhinitis. Cases of chronic sinusitis can be sharply and accurately differentiated into two etiologic varieties: the pure infective sinusitis due generally to an anaerobic type of streptococcus, and the allergic sinusitis due to an underlying nasal allergy with secondary infection. The best therapeutic results will follow only when the infection and the allergy are treated simultaneously, and this can be accomplished best by the rhinologist who makes his own allergic studies.

II. Methods of Diagnosis of Nasal Allergy

The first step in the diagnosis of nasal allergy is to suspect its presence from the character of the symptoms and appearance of the nasal mucosa. Remembering that the nasal mucosa normally has a remarkable ability to throw off infections, chronicity of a nasal infection or prolongation of an ordinary head cold should always suggest an underlying allergy. There may be the classical allergic symptoms of sneezing, nasal blocking and watery nasal discharge, but more often patients with chronic nasal allergic rhinitis and sinusitis will complain only of nasal blocking and tenacious mucoid nasal or postnasal discharge. Particularly characteristic is the increased susceptibility to "head colds", and the tendency of these to last weeks or months, instead of days. Periodic headache, with or without nasal stuffiness, is a frequent and characteristic symptom in nasal allergy. The presence of other allergic symptoms should also point to the possibility of an underlying allergy in any chronic nasal or sinus infection. These other allergic symptoms are hay fever, asthma, gas and distress after eating, food idiosyncrasies, eczema and urticaria.

The typical allergic nasal mucosa is pale and edematous, with increased clear watery, or mucoid secretion. Nasal polypi are almost always on an allergic basis, and polypoid and hyperplastic changes in the turbinates are usually allergic in origin. The finding of thick tenacious mucopus filling the inferior meatus is very characteristic of a nasal allergy. In atypical cases, the nasal mucosa may be red instead of pale, with a tendency to dryness.

Examination of the nasal smear for eosinophiles is very helpful in diagnosing an allergic type of rhinitis, but several examinations may be necessary to demonstrate them. As the rhinologist becomes more familiar with the diagnosis of chronic allergic rhinitis, he will depend less on a positive nasal smear.

Having determined that the patient probably has a nasal allergy, it becomes necessary to determine the allergen or allergens; that is what substance or substances are causing the nasal symptoms. The methods of diagnosis at our disposal are:

1. The Allergic History. The history is one of the most valuable aids in diagnosing the specific allergen, and in our experience it is often more valuable than the skin tests. Where the history shows that the symptoms have a seasonal incidence, or are improved by a change of climate, a trip away from home, or a vacation from work, we may assume that we are dealing with an allergen inhaled from the

INHALANTS

H. DUST	1-10	+		
KAPOK	.001	—		
ORRIS	.001	—		
COTTON S.	.001	—		
FLAXSEED	.001	—		
PYRETH	1-100	—		
TOBACCO	1-10	—		
SILK W.	.001	—		
FEATHERS	.1	—		
RABBIT	.001	—		
DOG	.001	—		
CAT	.001	—		
GOAT	.1	—		
COW D.	.01	±		
CAMEL D.	.01	—		
WOOL	.1	±		
HOG D.	.001	—		
HORSE D.	.001	—		
HORSE S.	1-100	—		
GLUE	.0005	—		

Fig. 5.—Case of Dust Allergy, Miss C. E., age 12.

air, for the diet of the average person remains fairly constant. If the symptoms occur only during the pollinating season of certain plants, the diagnosis of the allergen is obvious. If the nasal symptoms are better in summer, worse with cold weather and in steam heat, worse indoors than out, worse when dust is stirred up, and improved by a trip to the south during the winter, house dust is probably the allergen regardless of the skin test (Fig. 5). When a food is responsible for nasal symptoms, these symptoms are usually not affected by season, climate or locality, and they are usually, but not always, associated with gas and distress after eating, or headaches.

An example of an allergy to house dust despite the negative skin test is shown in the case of Miss C. E., age 12.

Intradermal tests with the common inhalants were essentially negative (Fig. 5). The patient gave a history of chronic productive cough, chronic purulent nasal discharge from the age of 4; three attacks of pneumonia. She had been treated for sinusitis and bronchitis for four years by repeated bronchoscopic aspirations, intranasal antrotomy and repeated irrigations of the antrum, with considerable improvement in the cough and the nasal symptoms, but mucopurulent secretion was always obtained from the nose and bronchi. The patient spent seven weeks in a summer camp with complete disappear-

ance of productive cough and nasal discharge for the first time since early childhood. Bronchoscopic aspiration and irrigation of antra were both negative on her return home, but within three weeks the symptoms had returned, and pus was obtained from the bronchi and the antra. Despite the negative test to dust, treatment with autogenous dust extract resulted in almost complete relief from chronic bronchitis and sinusitis.

2. Skin Tests. The skin tests are a useful aid, but cannot be relied upon as a means of diagnosis. As a rule the skin tests are relatively accurate and reliable where the allergen is an inhalant, such as pollens, fungi, house dust, feathers, or orris root. In food allergies, skin tests are reliable in about 30 per cent of the cases, and in drug allergies, the skin tests are negative and cannot be used for diagnosis.

It is unnecessary to do a large number of skin tests in every case of nasal allergy. The average case of nasal allergy can be successfully diagnosed and the patient relieved of his symptoms in our experience with the use of 20 to 50 skin tests. It is the exceptional case that requires more than 50 tests. If the symptoms indicate an inhalant allergy, worse in winter, the patient is tested with 16 common inhalants. If the patient has pollinosis symptoms, the common pollens and fungi are tested for. If the symptoms suggest a food allergy, the patient checks the foods that he eats once a week or oftener, and he is tested for these foods. There seems little point in testing for a food the patient rarely or never eats.

To be of value, skin tests must be made with potent allergens, diluted properly. This means that extracts of fruits and vegetables will be used concentrated, or in a 1:10 dilution, whereas, some of the nuts, pollens and animal danders must be diluted 100 or 1000 times. It is essential that the skin tests be made and read by the correct technique. We use the intradermal tests exclusively, finding that they can be made as quickly and with less pain than the scratch tests, and that they are more accurate and reliable for diagnosis than the scratch tests. If used in proper dilutions and with proper technique, the intradermal tests are relatively safe, if one remembers always that serious, and even fatal, reactions can occur from any skin test, either scratch or intradermal, but that such reactions, for practical purposes, are always in asthmatics. In testing asthmatics, we must be on guard against possible systemic reactions by testing with only a few allergens at one time, sometimes not more than one or two; by being certain to use sufficiently diluted allergens, particularly if the history points to that substance as the probable cause of symptoms, as for example,

horse dander. Adrenalin for hypodermic injection, and a tourniquet to be applied above the test should always be in the room when skin tests or injections for treatment are being done. At the first sign of a systemic reaction, consisting of a cough, wheeze or hives, the tourniquet should be applied. Three-tenths (0.3) cc. of adrenalin should be injected into the site of the skin test, and three-tenths (0.3) cc. injected subcutaneously into the other arm. The tourniquet, of course, must be released periodically, according to the patient's symptoms.

To make the reading of skin tests uniform, the same area and type of skin should be used, namely the outer surface of the upper arm. Tan and sunburn will greatly lessen intensity of the skin reaction, and should be allowed to subside before testing. An area of skin which has reacted positively will be refractory for several days, so that the same area should not be retested sooner than three days. Because a strongly positive test will cause adjacent tests to appear falsely positive, the tests should be spaced at least one inch apart in a horizontal direction, one and one-half inches in a vertical direction. With this spacing, twelve tests can be made on the outer surfaces of each upper arm of an adult. The tests should be read at the height of the wheal formation, which will vary between 10 and 15 minutes. The same amount of material is injected in each test, approximately 0.01 cc. from a tuberculin syringe with a 27 gauge needle. The syringe is held with the bevel toward the skin. The skin is engaged with the tip of the needle, the syringe depressed so that the bevel is pressed tightly against the skin, and enough material is injected to cause a definite, but tiny wheal, the size of the diameter of a match head. Properly done, this technique is virtually painless. The diluting fluid used in preparing the allergens is injected in the same amount at the top of the arm as a control. The tests are graded according to the size of the wheal, from negative to four plus, the latter being a wheal with pseudopods. We pay very little attention to erythema in judging the reaction.

Again we repeat, the skin tests must not be relied upon as the means of diagnosis. There are too many people going around with a long list of foods which they shun on the basis of skin tests of doubtful significance. With experience, we learn to attach more significance to a slightly positive test to a food when all the rest of the tests are negative, than to a strongly positive test, when there are many strong positives. We also learn that a positive test to certain foods, such as chocolate, coffee, wheat, milk and egg is more apt to be significant than an equally positive test to a fruit or vegetable.

BASIC DIET

May have—

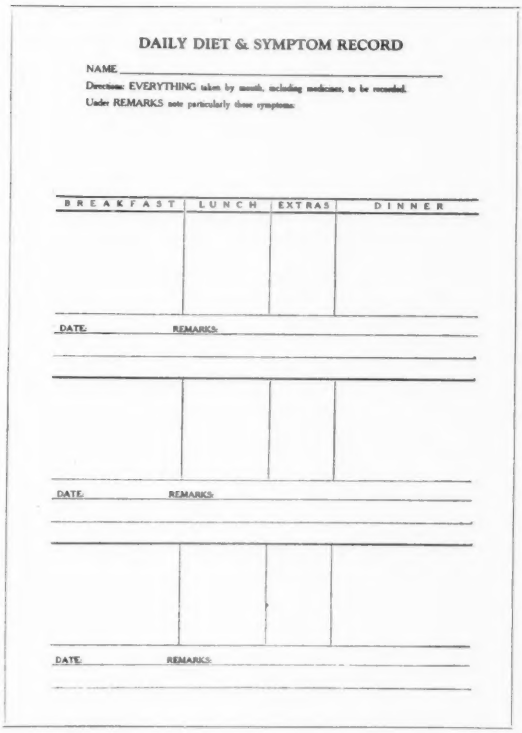
Meat	well cooked beef and lamb
Fruits	cooked figs canned pears banana
Cereal	oatmeal
Vegetables	lettuce spinach beets turnips carrots endive white potato squash
Butter	
Beverages	tea
Desserts	jello tapioca plain sugar candy

NOTE: Wheat, eggs, milk in all forms are excluded.

PREPARATION: May use salt, olive oil, maple syrup and sugar.

3. Elimination Diet. Since skin tests for food are unreliable in the majority of food allergies, we must rely largely upon elimination diets for diagnosis. We find that a Basic Diet, modified from Hansel, is the most practical. This diet eliminates all of the foods commonly causing allergy, but leaves the individual enough to maintain his strength, if not his weight, for the trial period of one to two weeks. This length of time may be necessary because a food will continue to be found in the circulation several days after it has been eaten. It is essential that the patient keep diet cards (Fig. 6), on which he records everything taken by mouth, because even the most intelligent patient after the most careful instruction, may fail to follow the diet correctly the first time.

4. The Therapeutic Test. In the final analysis, the proof of a nasal allergy depends upon the therapeutic test. When removal of



DAILY DIET & SYMPTOM RECORD

NAME _____

Directions: EVERYTHING taken by mouth, including medicines, to be recorded.
Under REMARKS note particularly these symptoms:

BREAKFAST	LUNCH	EXTRAS	DINNER

DATE: REMARKS:

DATE: REMARKS:

DATE: REMARKS:

Fig. 6.

a specific substance or treatment with an extract of it results in recovery from the symptoms, and when exposure to the substance again produces the symptoms, we may regard this as the allergen responsible for the trouble. A frequent mistake is to rely upon skin tests, rather than the therapeutic test for diagnosis. *The skin tests must be regarded as an aid, but not the means to diagnosis.*

SUMMARY

From the character of the patient's symptoms, the appearance of the nasal mucosa, and the finding of eosinophiles in the nasal smear, a nasal allergy is suspected. To find the offending allergen we have the patient's history, the skin tests, the elimination diet, and the

therapeutic test. In the last analysis, the proof of an allergen depends upon the therapeutic test: recovery from symptoms when the specific substance is removed, and recurrence of symptoms when the patient is again exposed to the substance. The skin tests must be regarded merely as an aid, and not as the sole means of diagnosis.

III. Treatment of Nasal Allergy

If we exclude pollinosis (hay fever), house dust is a major factor in at least 90 per cent of all cases of chronic nasal allergy that we see. The treatment of nasal allergy due to house dust sensitivity by means of injections of house dust extract gives extraordinarily satisfactory results, provided the proper dosage is used. We do not know the mechanism underlying the improvement of symptoms that follows subcutaneous or intradermal injections of the allergen. We do know that extremely minute doses often give better results than larger doses, and that it is not always necessary to build the doses up to get complete relief. In treating a case of house dust sensitivity, the chief problem is to determine the optimum dose for the individual. This varies greatly; from 0.05 cc. of 1:100,000,000 dilution to 0.2 cc. of 1:1,000 dilution. We are now using considerably smaller doses than formerly, and only rarely do we give an extract as concentrated as 1:1,000. As a general rule, patients with asthma or dermatitis due to house dust sensitivity will respond better to the higher dilutions, whereas, patients with mild nasal symptoms often respond better to 1:10,000 dilution. Where the intradermal skin test results in marked improvement in symptoms, we usually begin with 0.05 of 1:10,000 dilution. Where skin tests result in a flare-up of symptoms, or no improvement, we begin with 1:1,000,000 or 1:10,000,000 dilution. If the patient experiences a flare-up in symptoms following an injection, a more dilute solution is used the next time.

The injections are given subcutaneously, once a week, and the dose is slowly increased, or decreased in case of a reaction, until the symptoms are relieved. That size dose is then regarded as the optimum for that patient, and it is repeated once every three weeks until warm weather arrives. In some cases the symptoms will recur before the three weeks are up, and the maintenance dose will need to be given more often. Should the symptoms recur during the summer, and particularly with the coming of cold weather in fall, the injections are resumed beginning usually with about one-quarter size of the last dose, and building up the dose until symptoms are again

Schedule of Dust Extract Dosage

1. Cases of typical mild chronic rhinitis due to dust

   Symptoms:
   Nasal blocking, occasional sneezing, postnasal discharge, frequent and prolonged head colds, lasting through the winter, clearing up in warm weather.

   Slight to moderate positive skin reaction, followed by improvement after skin test.

   Dosage:
   Begin with 1:100,000 dilution, 0.05 cc., double the dose until symptoms are relieved. Maintenance dose will be between 0.2 cc. of 1:100,000 and 0.2 cc. of 1:10,000 dilution.

2. Cases of severe chronic rhinitis with marked polyposis.

   Symptoms:
   Excessive sneezing, asthma, eczema of exposed surfaces, worse in winter usually with strongly positive skin reaction. May be unchanged or worse following skin test.

   Dosage:
   Begin with 0.05 cc. of 1:1,000,000 or 1:10,000,000 dilution, increasing dosage slowly watching for any reaction, in which case dose is dropped. Maintenance dose is anywhere between 0.05 of 1:100,000,000 up to 0.2 cc. of 1:1,000,000 dilution.

   All doses are given once a week until symptoms are relieved. Then once every three weeks, with a few exceptions requiring more frequent maintenance doses.

relieved. In severe and stubborn cases of house dust allergy, the patient should carry out "dust precautions" in his home, and particularly in his bedroom, where he spends a large part of his life.

The treatment of food allergies consists of removing the allergic foods from the diet. Injections of food extract have not proven satisfactory in the treatment of food allergies; however, if a patient remains away from a food for several years, he may lose a considerable part of his sensitivity to it, so that he may be able to resume the food in moderation without trouble. But, when an allergic patient modifies his diet by substituting other foods for allergic ones, he may acquire new sensitivities to some of the new foods which he is taking in increased quantities.

In treating allergies we must keep in mind that the allergic patient is a person who, for some as yet unknown reason, becomes hy-

Preparation For A Dust Free Bedroom

Approximately one third of a person's life is spent in bed; therefore the elimination of dust in the bedroom is of great importance.

No clothes should be left hanging in the room. The closet door must be kept closed.

No draperies, except for washable curtains, which should be washed once a month.

No rugs, except washable rag rugs. These are to be washed once a month.

No overstuffed furniture; only bare chairs.

Mattress and pillows on bed should be covered with dust proof coverings.

Comforter should be of cotton, rather than down.

Blankets should be of cotton, rather than wool. If wool blankets are used, they should be washed at least four times a year, and should be well covered by a sheet on top, as well as beneath.

Floors should be waxed or varnished, and cleaned once a day with an oil mop.

Moldings around doors and windows, and furniture including the bed, and bed springs should be wiped with a moist or oiled cloth once a week.

All cleaning should be done by a person other than patient. If done by the patient a mask must be worn over the nose and the mouth.

persensitive to foreign substances which he breathes, eats or touches. As a rule, the allergic patient is sensitive to a number of substances, but one or two of these will be chiefly responsible for his symptoms, so that when these one or two substances are removed, or he is treated for them, his symptoms will be relieved. Such a patient is considered to be in "allergic balance". When in "allergic balance" small doses of substances to which the patient is mildly allergic will produce no symptoms. The patient can be thrown out of "allergic balance" by contact with a substance to which he is highly allergic, or by large amounts of substances to which he is mildly allergic. Certain extraneous factors, such as nervous fatigue, worry, and infection may tend to throw a patient out of "allergic balance". In a sense, an allergic patient is never cured, because he still retains sensitivities to certain foreign substances, and at any time he may be thrown out of "allergic balance" by one of the above-mentioned factors.

In the treatment of nasal allergy the presence of complicating infection of the nasal mucosa and sinuses is an important factor which

Results Obtained In Patients Treated For Allergy Jan. & Feb. 1944

Chronic Rhinitis	Symptoms relieved—pt. dischgd.	17
	Symptoms relieved—pt. on maintenance	21
	Symptoms improved—not controlled	12
	Therapeutic failures	6
		56
Headaches	Symptoms relieved—pt. dischgd.	1
	Symptoms relieved—Pt. on maintenance	2
	Symptoms improved—not controlled	3
	Therapeutic failures	0
		6
Ménière's Disease	Symptoms relieved—pt. dischgd.	2
	Symptoms relieved—pt. on maintenance	1
	Symptoms improved—not controlled	1
	Therapeutic failures	0
	Did not return	1
		5
Eczema	Symptoms relieved—pt. on maintenance	1
Deafness	Symptoms relieved—pt. dischgd.	0
	Symptoms relieved—pt. on maintenance	2
	Symptoms improved—not controlled	0
	Therapeutic failures	1
		3
Chronic Sinusitis	Symptoms relieved—pt. dischgd.	3
	Symptoms relieved—pt. on maintenance	9
	Symptoms improved—not controlled	7
	Therapeutic failures	1
		20
Secretory Otitis Media	Symptoms relieved—pt. dischgd.	0
	Symptoms relieved—pt. on maintenance	1
	Symptoms improved—not controlled	13
	Therapeutic failures	0
	Did not return	1
		15
Bronchial Asthma	Symptoms relieved—pt. dischgd.	1
	Symptoms relieved—pt. on maintenance	3
	Symptoms improved—not controlled	3
	Therapeutic failures	0
	Did not return	1
		8

Results Obtained In Patients Treated For Allergy Jan. & Feb. 1944
(*Continued*)

External Otitis	Symptoms improved—not controlled	1
Nasal Polyps	Symptoms relieved—pt. on maintenance	1
Frequent Colds	Symptoms improved—pt. on maintenance	2
Chronic Laryngitis	Symptoms relieved—pt. on maintenance	2

must not be overlooked. In order to restore the patient to "allergic balance", and to relieve his symptoms completely, the infection as well as the allergies must be recognized, and treated. It is entirely impractical for the average patient to make repeated visits to two different doctors for his chronic nasal and sinus condition. It is for this reason that I believe every rhinologist must do his own allergic studies to get the best therapeutic results.

An analysis of the patients treated for nasal allergy during the two winter months of January and February 1944 showed the following: Of the 121 patients seen, 56, or nearly half suffered from a chronic rhinitis, 20, or approximately 1 out of 6 suffered from chronic sinusitis, 15 from a secretory otitis media, 6 from headache, 8 from bronchial asthma, 5 from Ménière's disease, 3 from primary nerve deafness, believed to be of allergic origin, 2 from eczema, 2 from frequent head colds, 2 from chronic laryngitis, and 1 from nasal polypi. The results obtained in these cases are summarized below.

SUMMARY

A large proportion of the patients seen in office practice of otolaryngology suffer from chronic nasal symptoms on an allergic basis, frequently complicated by superimposed infection. The best therapeutic results can be attained only when both factors are treated simultaneously, and this can be done best by the rhinologist who makes his own allergic studies. The history, the stained nasal smear, skin tests, elimination diet, and therapeutic test are the methods at our disposal in the diagnosis of a nasal allergy. Sensitivity to house dust is a major factor in most of the nasal allergy encountered in rhinological practice. Therapeutic response to injections of house dust extract is very satisfactory in the great majority of cases.

122 South Michigan Blvd.

IV

POST-WAR DEAFNESS, A CHALLENGE TO THE OTOLOGIST

BERNARD J. MCMAHON, M.D.

ST. LOUIS, MO.

This challenge to the otologist is not new. That he has failed to adequately meet the challenge may be judged by this statement of Guild[1] in 1918, writing on war deafness and its prevention, "the evidence at hand clearly indicates that the search for a prophylactic measure to prevent detonation deafness must be directed toward the prevention of injurious air-conduction," compared with that of Taylor[2] in 1943 in which he says, "it seems that the present emergency has stimulated an interest in the subject of the prevention of war deafness, which has been more or less latent since the close of the last war. . . . It is noted that in this third year of the second World War, interest in this phase of preventive medicine is being manifested, and progress in prevention is beginning to be made."

It is true that a great deal has been written on the subject of traumatic deafness and its prevention during this interval, but the sum and substance of the conclusions is that traumatic deafness of whatever origin is characterized by a loss of high tones, and something should be done to protect the ears of the soldier and the industrial worker in the future. Nevertheless, it has taken another World War to bring us to the realization that no real progress has been made in helping us to meet these responsibilities as otologists. The accepted and generally used ear defender is still a cotton plug, with or without vaseline, inserted into the aural canal.

The destructive lesions resulting from explosives are in direct proportion to their proximity to the individual. Such may occur in the open field or on board ship, on the open deck, below decks or in gun turrets, from bomb explosions, or from large guns.

In contrast to this immediate injury to the ear is that suffered from the constant repetition of an intolerable noise, such as is ex-

Read at the Thirty-Eighth Annual Meeting of the Southern Medical Association at St. Louis, Missouri, November 13, 1944.

From the Department of Otolaryngology, St. Louis University School of Medicine, St. Louis, Missouri.

perienced by the individual exposed to the noises associated with Diesel engines, motors in airplanes and in tanks, and small gun fire. This may be termed a debilitative insult to the eighth nerve, which eventually amounts to a distinct injury after a certain length of time. This injury is reversible within limits, since recovery is known to occur, as shown by the audiometric curve, after a period of rest sufficient to permit the nerve to regenerate physiologically. Colonel Willard Machle[3] immediately of Fort Knox, Kentucky, has reported that after a day of exposure to steady firing, the instructors at a gunnery school consistently showed an audiometric curve characterized by a drop in the high tones and a recovery to normal after several hours of rest. This fact has likewise been demonstrated by many other observers. However, after repeated and prolonged exposures the patient does not recover his high tone loss, and in addition there are certain isolated tones in the lower ranges which are depressed. While we accept the statement that the locale of high tone perception is about the basal turn of the cochlea, the most vulnerable portion because of its proximity to the round and oval windows, we may be at a loss to explain the vulnerability of an isolated area in the organ of Corti, unless it is caused by a discrete hemorrhage in the cochlea[4] or by repeated "jarring" of the entire cochlea by the force of the explosions, since a direct extension of the degenerative process would result in a progressive absence of perception of sound toward the lower end of the scale.

Crowe, Guild and Polvogt,[5] after careful microscopic studies of the histological changes which occurred in the cochleae of 79 human ears which showed a high tone loss during life, offered the following classical description of the pathological changes which are found:

> "Total atrophy of the nerve is extensive in some cases, in others it is localized. There is always total atrophy of the organ of Corti in the regions in which the nerve has entirely disappeared. . . . The transition from total absence of nerve fibres to normal nerve within a space of 1 mm. or less is often seen in this group in which there is an 'abrupt' and marked functional loss of hearing for tones an octave apart. . . .The most distinctive histological lesion in this group of ears with an abrupt high tone loss is atrophy of the organ of Corti at some level above the last 2 mm. of the basal turn. The atrophy of the organ of Corti, however, is always limited to the basal turn. . . . The extent of the atrophic area of the organ of Corti is not related to age . . . there is no evidence that a damaged organ of Corti regenerates, so it must be due to chance alone that so many with this type of lesion are among the younger patients. Further study of this point may lead to a suggestion of the etiology. . . . We have found extensive atrophy of the organ of Corti in but one female. The lesion is certainly more common in males. . . . Whether the total atrophy of the organ of Corti be extensive or localized, the external sulcus cells are always atrophic in the same area."

In considering post-war deafness, we are inclined to think only in terms of military aural casualties, while similar civilian casu-

alties will undoubtedly occur in great numbers. Unfortunately, very little statistical data on the incidence of industrial deafness will be available for obvious reasons of policy on the part of industrial concerns. It has been proven[6] that when animals are exposed to a sound of a certain pitch for various lengths of time, degeneration of the cochlea occurs. This was found to be caused particularly by notes of high pitch.

In industrial plants these prerequisites for cochlear damage are perfectly met. There, individuals are exposed to irritating sounds of great intensity, both constant and intermittent, for eight or more hours a day, for many months, without a remission to allow the cochlea to regenerate. We may expect men who have been accustomed to such sounds to have developed a type of conditioned reflex in the intra-aural muscles, which may protect the cochlea to some extent, but when there are thousands of women taken out of quiet environments and placed under similar noisy conditions, we cannot hope for such protective mechanism to be established in so short a time.

The hum of the motors in which there are undetectable and injurious high-pitched notes,[7] the screech of the milling machine, the clatter of the air hammer and the punch press, and the terrific crash of the gigantic metal press will undoubtedly take their toll of human hearing. Protective devices which should be worn in the ears do not suffice, not because of their inefficiency but because of the human element. The workers do not wear them consistently because of the immediate inconvenience of not being able to hear clearly, because of carelessness, and because of the conviction that such sounds to which they have become accustomed are not injurious to their hearing.

We are aware of the fact that many thousands of soldiers have been exposed to and have actually contracted malaria in various tropical theatres of war. This has obviously necessitated the administration of large amounts of quinine, and we might conjecture on the possibility of deafness resulting from the degenerative changes in the cochlea which are known to be caused by quinine. Yet, on inquiry from authoritative sources, we are told that there has been a negligible amount of ear injury from this cause. A favorable factor has undoubtedly been the availability of atabrin which has consistently been used as a suppressive of the parasite over greater lengths of time, while quinine has been used largely in the first three days of treatment, or as an abortive agent. In those patients, receiving the larger doses, who showed hearing defects, the quinine was reduced in amount or dis-

continued entirely. This is most heartening, and a tribute to the careful supervision which has been exercised in the administration of the drug by our fellow physicians in the armed services.

The accurate diagnosis of the various types of deafness will, as always, be most important. While otologists are becoming increasingly aware of the impracticability of the pure tone audiometer as a measure of the degree of usable hearing, it will continue to hold the most important place in our armamentarium until a more accurate spoken voice audiometer is devised and made available to the profession. Time does not permit of a discussion of the relative technical values of these two instruments, but we feel that each will have its place, together with the tuning forks, in assisting the otologist to properly diagnose deafness. We do not feel that an absolutely soundproof room is a necessity, though it is a valuable adjunct, since the determination of usable hearing should to some extent be ascertained in an average environment, or in a relatively soundproof room from which the unusual sounds can be excluded.

We must expect five types of deafness: (1) Conductive—from destruction and occlusion of the aural canal or from destruction of the middle ear mechanism, with subsequent adhesions; (2) Receptive—from injury to or destruction of the cochlea or the eighth nerve; (3) Mixed—from a combination of the above mentioned factors; (4) Functional; (5) Combined organic and functional.

The destructive injuries to the aural canal and the middle ear resulting in a conductive deafness should be corrected by adequate plastic surgery of such malformations as have taken place in the healing process, in order to enable us to evaluate the middle ear pathology and its influence upon the deafness. Thus such middle ear pathology as is amenable to treatment will be rendered accessible.

The cases of receptive deafness will probably be our most pressing problem, not only because of the variability in the degree of deafness and the insidiousness of its progress, but also because it will be found in civilian as well as in military aural casualties. A cochlea which has sustained an injury, whether instantaneous or gradual, will undergo certain changes affecting its function, and we have no absolute certainty of the extent of this injury, or whether it will progress or regress. Nor have we any way of estimating beforehand the rate of progression or regression. We apparently have been remiss in the past in not observing similar cases occurring in civilian practice, at regular intervals over long periods of time, except in a few research centers; otherwise more information on a larger num-

ber of cases would be obtainable from the literature. One of the most illuminating series of observations reported was that of McCoy,[8] in which he demonstrated a progressive high tone loss in a series of audiometric tests of shipyard workers on whom audiograms had been found to be normal at the time of their employment. These men were employed chiefly as operators of riveting and chipping hammers in confined areas.

In the mixed type of deafness, the exact delineation of the proportion of conductive and receptive deafness can only be ascertained by the most careful diagnostic procedures. These should be directed towards the elimination of obstructive factors first, as many will be remediable, and followed by an evaluation of the remaining deafness. In this regard, it is stated that if the membrana tympani is ruptured by the explosion, there is less trauma to the cochlea, because the force is dissipated instead of being transmitted in full along the ossicular chain to the footplate of the stapes.

Functional deafness, including hysterical deafness and malingering will undoubtedly be brought to our attention. The decision on the method of handling such cases will rest between the otologist and the psychiatrist, with each doing his part in the rehabilitation of the patient. These will not differ materially from most such cases encountered in civilian practice, except that the factor of maladjustment and unwillingness or inability to face the reality of the return to a not-too-promising industrial or domestic life will probably play a most important part.

Deafness resulting from combined functional and organic causes will offer greater difficulties of solution, especially in the management of the long period of adaptation of the patient to his handicap, and the adaptation of family and friends to the physically and mentally handicapped patient. It is in this type of case that the ingenuity of the otologist will be called upon to bolster the morale of the patient who is becoming accustomed to wearing a hearing aid; at the same time he should make periodic audiometric tests to determine the course of the deafness, and should reassure the patient that everything is being done to help him physically and psychologically, even though it is found necessary to resort to the aid of a psychiatrist. Likewise, the family, the wife or husband particularly, should be made to realize that the problem of readjustment lies in their hands also, and that the social life of the patient should be so modified that the handicap will be less keenly felt. Advice should also be given to the patient and the employer in the selection of an employment least likely to

aggravate the debilitated hearing mechanism, because of our uncertain knowledge of the ability of the cochlea to regenerate, since the vulnerability and the powers of recovery of the human cochlea and the eighth nerve are undoubtedly highly individual.

In the report of the Committee on the Conservation of Hearing of the American Academy of Ophthalmology and Otolaryngology, in October 1944, a carefully planned and most commendable outline was set forth on the "Audiometric Procedures in the Administration of a Program for the Conservation of Hearing of School Children." While this plan is equally applicable to the care of the deafened of any age from the standpoint of municipal and state agencies, it does not necessarily apply in its entirety to the care of the patient by individual clinics and otologists. It would therefore be expedient to modify some of these suggestions in accordance with the problem of each patient.

The acceptance of this challenge by the trained otologist should therefore be manifested by the formulation and execution of a definite plan. This plan should be applicable not only to private practice but to clinic practice as well. His services should be offered in clinics in his locality, preferably in affiliation with a teaching institution which has the equipment and the personnel available to properly diagnose and treat deafness of all types.

It should be the function of such clinics to give unbiased advice in the selection of proper hearing aids after a careful detailed history, both personal and familial, has been taken, a thorough examination of the ears, nose and throat has been done, and the combined evidence evaluated.

Heretofore there have been too many factors beyond our interest and control. Not the least of these has been the campaign of high-powered salesmanship by the manufacturers of hearing aids. I shall not devote valuable time to a repetition of the disadvantages to the patient of this type of propaganda, nor to any suggestions for the correction of this abuse. Since our chief concern is the good of the patient, and the chief concern of the manufacturers of hearing aids is the marketing of their product, it is obvious who may be the beneficiary. The patient himself may not realize the fallacy of the situation, and may be convinced that he is doing all that can be done to benefit his deafness. It is, therefore, the duty of the otologist to educate the public towards a better understanding of what may be done in a scientific way for the improvement of hearing.

A single audiometric test, while of great value in itself, should be repeated once and possibly twice at the same time early in the morning to insure greater accuracy as the patient becomes more accustomed to the procedure. When a fatigue factor is in question, the test should be repeated later in the afternoon of the two or three respective days by the same person. When accurate phonographic audiometers are available, these tests should be coupled with those of the pure tone audiometer. If a hearing aid is prescribed, it should be checked and the audiometric tests repeated at least every two months in order to detect any change in hearing acuity and advise the patient accordingly. If no change is discernible after six months, the examination should be repeated every six months after that. The patient should be apprised of the possibility of a change in his hearing occurring with subsequent nasal infections and instructed to report for a re-examination if such do occur.

This type of study of patients with past-war deafness is essential if we are to acquire a sufficient amount of valuable scientific clinical data on the progress of traumatic deafness to be eventually able to be of greater assistance to our aural casualties.

Before closing, a word about the medicinal treatment of deafness is in order. A great many of us have prescribed vitamins, especially of the B group, with the idea of stimulating the recovery of the debilitated eighth nerve. There is no scientific evidence to substantiate this hypothesis. However, if such medication will improve the general state of the patient's health, the administration is justifiable. By the same token thyroid medication is indicated in individuals with a lowered basal metabolic rate, the rationale being that the circulation in the internal ear will be stimulated, accumulated waste products of a toxic nature will be carried away and the internal ear and the eighth nerve be permitted to function more normally, thus establishing an optimum condition for regeneration of the neural mechanism if it is possible for it to occur. The added factor of the feeling of well-being and the increase in alertness of the patient will also contribute to the functional improvement in his hearing.

The responsibility for the care of the post-war aural casualties rests with us and we must not lose sight of this fact, that to the extent that we are delinquent in our duty to these, to that extent shall we ourselves cut the pattern for socialized, government-controlled medicine in our post-war world.

SUMMARY

1. There will undoubtedly occur during and after the present war many cases of traumatic and percussion deafness, both among the military personnel and among civilian workers in war defense plants.

2. The careful diagnosis, advice and treatment is the responsibility of the otologist.

3. The treatment should not only be surgical if indicated, but also supportive and psychological, in order to help these aural casualties find and adjust themselves to a successful and happy place in the post-war world.

MISSOURI THEATRE BUILDING.

REFERENCES

1. Guild, S. R.: War Deafness and Its Prevention, J. Lab. and Clin. Med. 2:849, 1918.

2. Taylor, H. M.: Traumatic Deafness; Problems of Prevention, Laryngoscope 54:362, 1943.

3. Machle, Willard: Effect of Gun Blast Upon Hearing, Read before The Amer. Acad. of Ophthal. and Otolar., Chicago, Ill., Oct. 8, 1944.

4. Groves, W. E.: Otologic Observations in Trauma of the Head, Arch. Otolaryng. 8:36, 1918.

5. Crowe, S. J., Guild, S. R., and Polvogt, L. M.: Observations on the Pathology of High Tone Deafness, Bull. Johns Hopkins Hosp. 54:315, 1934.

6. Horton, G. P.: An Experimental Study of Stimulation Deafness in Guinea Pigs, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 44:252, 1935.

7. Lurie, M. H.: War and Deafness, Connecticut State M. J. 7:763, 1943.

8. McCoy, D. A.: The Industrial Noise Hazard, Arch. Otolaryng. 39:327, 1944.

V

AEROSINUSITIS—A RÉSUMÉ

COLONEL PAUL A. CAMPBELL, M.C.

RANDOLPH FIELD, TEXAS

As expansion of the activities of man led him to explore the higher reaches of the atmosphere, the depths of the sea, and the earth, the effects of barometric pressure variation upon the closed gas-and-air-containing cavities of the body became more and more evident, and consequently the subject of more and more investigative effort.

Until high altitude flight became somewhat common, very few persons had been subjected to great barometric pressure change. The numbers of aviators, deep sea divers, caisson workers, and those in other occupations requiring exposure to pressure changes were never great. Consequently, interest in pressure effects was limited to a few investigators, who for the most part confined themselves to the more spectacular effects—namely, decompression sickness and anoxia.

World War II, however, produced an incentive for mass high altitude flights, as the man "on top" was demonstrated to have great tactical advantage. Furthermore, flight at high altitude was proven to have certain economical implications, for the simple reason that thin air decreases the friction to which an airfoil is subjected. Consequently, air speed for any given power output is greater. In fact, at 35,000 feet it is some 70 per cent greater than at ground level.

Over-the-weather flights are also of great advantage. Weather is still the Nemesis of the airman, and he is willing to sacrifice much in the way of comfort to avoid it.

Simulated altitude flight by means of decompression or pressure chambers has been of interest to research groups since the middle of the nineteenth century. However, mass employment of simulated flight in the indoctrination and classification of flying personnel awaited the Altitude Training Program, one of the outstanding achievements of World War II. This procedure consists of a simu-

---

From the Research Laboratory of the Army Air Forces School of Aviation Medicine, Randolph Field, Texas.

Read before the Sixty-Sixth Annual Meeting of the American Laryngological Association, New York, N. Y., June 7-8, 1944.

lated flight to high altitude for the purpose of acquainting flying personnel with oxygen equipment and indoctrinating them in the use of oxygen at altitude.

The magnitude of the program can be realized when one considers that 275,000 air crew members received this training in 1943.[1] When one adds this number to the large numbers of men participating in aerial flight, one may understand that at this time a considerable number of people are being subjected to extreme barometric pressure change—the environmental factor which in certain instances may lead to the development of aerosinusitis.

Historical Data: Paul Bert, in his monumental volume, "La Pression Barometrique,"[2] published in 1878, did not allude directly to the effect of barometric pressure variation upon the nasal accessory sinuses. However, he did refer to the experience of M. Cezanne, who reported that during caisson work his employees sometimes experienced an issue of blood from the nose and throat, and that at times headaches persisted for several days. Again, he quoted Babbington and Cuthbert as reporting in 1861 that some of their workers suffered from nosebleed and headaches. The first detailed description of the entity later to be known as aerosinusitis was written by Marchoux and Nepper[3] in 1919, who after experiments in a decompression chamber stated:

> "Our observations lead us to believe that great importance is to be attached to the integrity of the rhinopharyngeal mucosa. Any obstruction which prevents the rapid establishment of pressure equilibrium between the cranial air spaces and the outside atmosphere involves the possibility of a series of misfortunes in which pain is one of the least and syncope one of the most serious. All become more marked as the pilot approaches land and at a time when he needs his faculties for a safe landing. While the atmospheric pressure decreases by 10 cm. of mercury between sea level and an altitude of 1,000 meters, it does not vary more than 4 cm. between 6,000 and 7,000 meters. It is therefore in the lower layers of the atmosphere that the defect is most felt."

Since that statement, several treatises concerning the effects of barotrauma on the sinuses have been published by various investigators, including Behnke and Willmon,[4] Brouwer,[5] Pastore,[6] Von Diringshofen,[7] Hermann,[8, 9] Salem,[10] and this observer.[11, 12]

Definition. Aerosinusitis[11] is an acute or chronic inflammation of one or more of the nasal accessory sinuses produced by a barometric pressure difference between the air or gas inside the sinus and that of the surrounding atmosphere. It is commonly characterized by congestion and inflammation of the lining structures. Pain over the area of the sinuses is usually present. Mucosal or submucosal hemorrhage

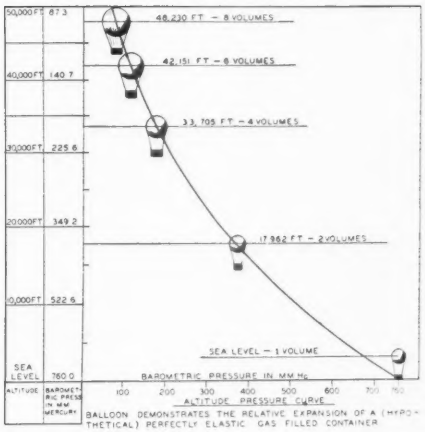
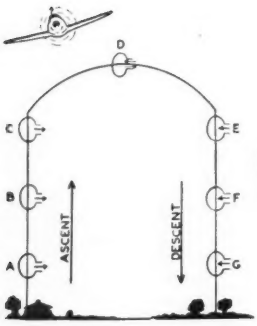

50,000 FT 87.3
40,000 FT 140.7
30,000 FT 225.6
20,000 FT 349.2
0,000 FT 522.6
SEA LEVEL 760.0
ALTITUDE
BAROMETRIC PRESS IN MM MERCURY
48,230 FT — 8 VOLUMES
42,151 FT — 6 VOLUMES
33,705 FT — 4 VOLUMES
17,962 FT — 2 VOLUMES
SEA LEVEL — 1 VOLUME
BAROMETRIC PRESSURE IN MM Hg
100 200 300 400 500 600 700 760
ALTITUDE PRESSURE CURVE
BALLOON DEMONSTRATES THE RELATIVE EXPANSION OF A (HYPOTHETICAL) PERFECTLY ELASTIC GAS FILLED CONTAINER

Fig. 1

may occur. The condition may at times result in temporary or permanent change in the mucous membranes lining the sinuses, depending upon the amount of barotrauma involved.

Incidence. Aerosinusitis, in terms of an entire population, is a very rare condition. In terms of that portion of the population which is being exposed to extremes of barometric pressure change, either in simulated or actual flight or in deep sea diving, it is not so rare. In the Altitude Training Program, depending more or less upon the month of the year, the geographical position of the altitude chamber, and using residual pain over the sinuses as the criterion, the incidence has been reported to vary from practically none to three or four per cent of those making flights above 25,000 feet. In a series of 35,000 simulated flights, again using this extreme criterion, the incidence was one and nine-tenths per cent. In a similar series of 9,000 man flights, only five one-hundreds of one per cent presented sinus symptoms serious enough to require hospitalization. Statistics of actual aerial operations are still meager. From evidence arriving from the field, it would seem that the incidence is only a small fraction of that occurring in the Altitude Training Program. From the standpoint of anatomical incidence, the larger sinuses are more often involved than the smaller ones. Thus, the frontal and maxillary sinuses are the sites of predilection. It has been the experience



FLIGHTLIKE EXCURSION OF A RIGID CONTAINER (SUCH AS A SINUS) WITH COMMUNICATION TO OUTSIDE ATMOSPHERE

Fig. 2

of this observer that frontal sinuses are involved more often than the maxillary sinuses. However, Wright[13] reports the maxillary sinuses to be the site of greatest incidence in his series. In terms of seasonal influence, the incidence, as would be expected, more or less parallels that of infectious rhinopharyngitis.

Physical Considerations. The density of the atmosphere which surrounds the earth's surface decreases with altitude. This decrease is such that at about 18,000 feet the atmosphere is only half as dense as at sea level; about one-fourth as dense at 34,000 feet; and about one-eighth as dense at 48,000 feet. As can be seen diagrammatically in Fig. 1, the change is not linear per unit of altitude but curvilinear.

Any material moving from a region of greater density to a region of lesser density, as in ascent, expands until equilibrium is established between the forces acting inside the material and those outside.[12] In the opposite manner, movement from a region of lesser density to that of greater density produces contraction.

If a closed gas- or air-containing cavity is moved from a region of greater density to one of lesser density, as in ascent, it will expand. The degree of expansion is limited by the pressure differential between the air or gas inside the cavity and that outside, and also by the restraint afforded by the elasticity of the material which makes up the structure. On descent, the opposite effect occurs when the forces are applied in an opposite manner.

On the other hand, a cavity with an opening accessible to the outside is affected during ascent or descent only in that the contents

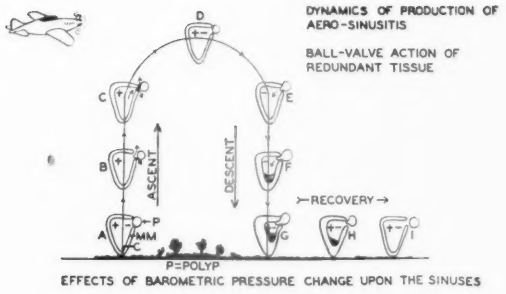


Fig. 3

of the cavity flow outward during ascent. On descent, material from the outside is drawn inward until equilibrium is established (Fig. 2).

Categories. As was pointed out, a cavity, such as a normal sinus with an unobstructed ostium, will during ascent allow its gaseous contents to move outward. On descent, the flow of air or gas is in the opposite direction. As this movement is not accompanied by any change in the structure of the sinus, no sensation is felt and no symptoms occur.[11]

This free flow of gaseous matter may be altered or prevented by two conditions. First of all, by the presence of pus, fluid, or similar substance covering the ostium; and second, by the obstruction of the ostium afforded by redundant tissue or anatomical deformity. The first situation has been categorized as *non-obstructive aerosinusitis*, and the second as *obstructive aerosinusitis*.

Non-Obstructive Aerosinusitis. If fluid, mucus, or pus covers the ostium of a sinus, changes in barometric pressure taking place during descent may press the material into the sinus. In this manner, uninfected sinus membranes may become infected. Infection is, however, a rather unusual occurrence unless the membranes have been severely barotraumatized, their natural immunity lowered, or they are insulted by virulent organisms.

A few such cases have been reported during the recent influenzal outbreak. The usual history has been that of a mild aerosinusitis following a simulated flight with apparent recovery after a period of 24 hours or so. However, from three to five days later, in certain instances, a rather serious purulent sinusitis has developed.

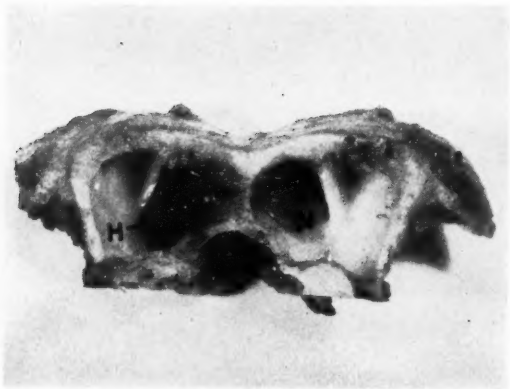


Fig. 4.—Gross appearance of hematoma, H, after sinuses were uncapped. N is normal control sinus which was not obstructed.

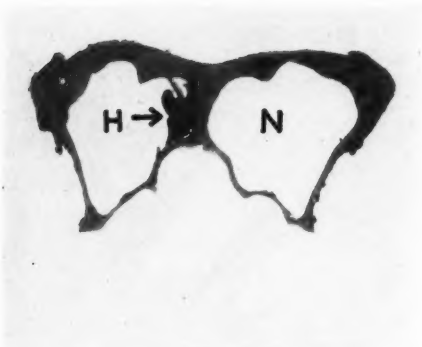


Fig. 5.—Transverse section through sinus showing hematoma, H. N is normal control sinus.

Obstructive Aerosinusitis. When the ostium of a sinus is blocked by swollen or redundant tissue or an anatomical deformity, a flap valve may be formed during pressure change, and the second category, that of obstructive aerosinusitis, results.

The forces' extent can best be understood by referring to a diagram illustrating the cycle of events resulting from an aerial excursion, be it simulated or real (Fig. 3).

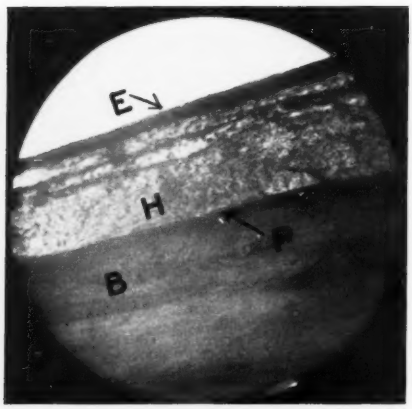
Let us consider the case of a maxillary sinus with a small polyp or bit of redundant tissue situated in such a manner that it can be pressed against the opening, thus forming a flap valve.

During ascent, the flow of the air or gaseous content is outward. Consequently, the flap is merely pushed aside and the pressure equalized. On descent, however, the situation is quite different as the heavier outside atmosphere presses the flap against the ostium, or as you may wish to consider it, the lighter inside air sucks the tissue into the ostium. An air-tight seal is thus formed. As descent is continued, a pressure differential develops between the air or gas inside the cavity and that outside. The result is that of a partial vacuum.

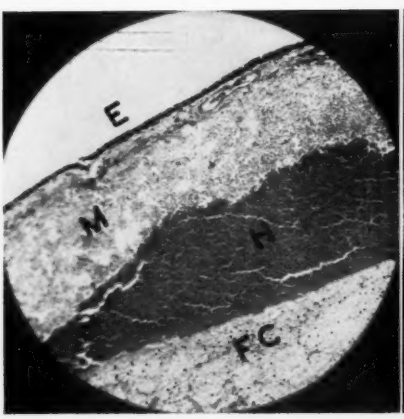
Equalization of forces acting and re-acting is a law of nature. The relative negative pressure inside the sinus can be equalized by any space-filling phenomenon. The most usual space-filling phenomena are swelling of the mucous membranes and fluid production, either into the cavity of the sinus or into or under the lining membranes. As soon as sufficient space is filled, the flap valve is released and recovery begins.

Pathological Changes Inside the Sinuses. At least two observers have examined directly the tissue alterations which have taken place following extreme barotrauma in man. Hermann[8, 9] has reported mucosal detachment and submucosal hematoma. Wright was forced to perform a Caldwell-Luc procedure upon a maxillary sinus which failed to respond to conservative management. He reported:[13]

"The periosteum of the canine fossa was somewhat bound down by fibrous reaction. The bone was thin and easily removed. On the opening of the sinus a bluish membrane bulged out under pressure. Incision of the membrane revealed a cavity entirely filled by many blood clots enclosed in membranous sacs and not free in the antral cavity. There was no free space in the antrum not filled by these rounded enclosed clots of old blood. There was a dark red color to all the structures. No blood could be evacuated without incising the membrane covering the clot. The entire membrane with the clots was removed and an intranasal opening was made. Microscopic examination showed the bleeding to have taken place in the subepithelial tissues of the mucous membrane."



A.



B.

C.

Fig. 6.—Hemorrhagic areas in the mucosa. E—Epithelium; M—Mucosa; H—Hemorrhage; FC—Fibrin clot; P—Periosteum; B—Bone.

In our laboratory we have produced aerosinusitis in a dog by the following procedure. The animal was placed under nembutal anesthesia and ascended to 28,000 feet in an altitude chamber. During ascent oxygen was administered as necessary. At 28,000 feet the chamber was leveled off and the area about the exit of the left naso-frontal duct was rather tightly packed with vaseline gauze. The animal was then returned to ground level.

After one hour, examination of the sinus in which the ostium had been obstructed disclosed a large hematoma extending upward from the internal orifice of the naso-frontal duct and occupying the principal part of the medial wall of the septum (Fig. 4).

There were also multiple small hemorrhages in the mucosa. The mucosa of the unobstructed sinus was normal. A transverse section of the sinus is seen in Fig. 5.

Microscopic studies showed that the small hemorrhages arose between the epithelium and the periosteum in highly vascularized areas of the mucosa (Fig. 6a, 6b, and 6c).

Presumably the large hematoma had a similar origin with the blood spreading along an indefinite line of cleavage between the dense fibrous tissue of the periosteum and the loose-textured connective tissue of the mucosa. At no point was the epithelial continuity of the mucosa broken. It is of interest that the superficial part of the hematoma was composed primarily of red blood cells while the deeper part consisted principally of coagulated plasma with networks of fibrin enmeshing scattered leukocytes (Fig. 7).

The experimental lesions were similar to those found in the surgically excised mucosa of a maxillary sinus of the patient seen by Wright and Boyd,[13] who kindly loaned me a section of the tissue demonstrated in Fig. 8.

Roentgenographic Findings. In view of the spectacular pathological changes noted at times, similar unusual roentgenographic findings would be expected. In many instances, especially in obstructive aerosinusitis, this is the case. Of most interest is the mucosal hematoma, illustrated in the following series of roentgenographic plates.

The condition illustrated followed an ascent to 38,000 feet in an altitude chamber, where the subject remained for a short period of time. Descent was then made at a rate of 6,000 feet per minute. When a level of 26,000 feet was reached, he suddenly noticed a sharp pain in his left supra-orbital region. The sensation was described as

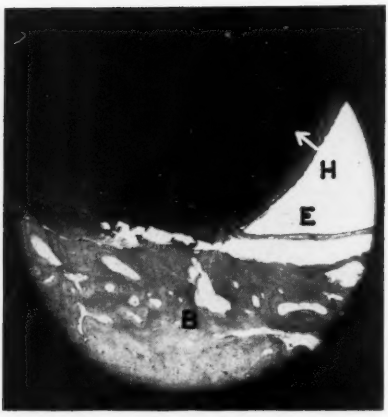


Fig. 7.—B, bone; E, reflected epithelium and mucosa; H, large hematoma.

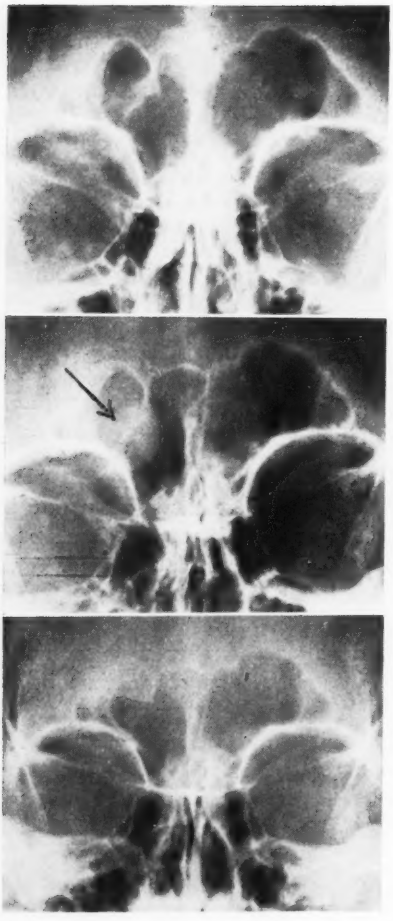
Fig. 8.—Mucosal hematoma from maxillary sinus of patient seen by Wright and Boyd.

feeling as though something had burst over and to the nasal side of his eye. The pain was relieved slowly by leveling off and then re-ascending about 1,000 feet. Descent was then begun again. During this descent it was necessary, because of pain, to level off five or six times before reaching ground level. There were no painful after-effects. However, an x-ray picture of his sinuses was taken (Fig. 9b) which, on comparison with a routine x-ray picture (Fig. 9a) taken a month before, demonstrated a large dome-shaped swelling of mucous membrane in the left frontal sinus, which had not been present at the time of the previous x-ray picture. Serial x-ray pictures taken subsequently demonstrated a slow resolution of the process with complete recovery about five months later (Fig. 9c). Roentgenographs shown in Fig. 10 are random roentgenographic studies illustrating various hematomas which have been seen in our laboratory.

The roentgenographic findings in non-obstructive aerosinusitis are in most instances not diagnostic, as the amount of fluid or pus pressed into the sinus is never large.

Diagnosis. Aerosinusitis must be differentiated from purulent and catarrhal sinusitis. The differentiation is usually simplified by the elicitation of a history of pain over one or more of the sinuses during or shortly after exposure to extensive barometric pressure change. In the more severe cases, especially those in which there has been stripping of the mucous membrane or mucosal or submucosal hematoma, the patient may describe the onset of the pain in such terms as "feeling as though a bee had stung him over the eye," as "being struck by a sharp piece of metal," or as "being struck in the head with a club or ball bat." In the less spectacular instances the pain may be described as having slowly developed after return to ground level. Epistaxis occurring during or after exposure to barotrauma is extremely suggestive. Roentgenologic plates disclosing opacity or a thickened lining membrane in the absence of previous sinusitis are strongly indicative. This is especially true if mucosal or submucosal hematomas are demonstrated. Leukocytosis and fever in most instances are absent in aerosinusitis. It must be borne in mind that aerosinusitis can occur in a sinus which is already infected, thereby altering the clinical picture of each of the entities.

Course. The milder cases of this manifestation of barotrauma are self-limited, resolution taking place in from a few hours to a few days. The more severe cases may run a course of from a few days to a few weeks. Absorption of mucosal or submucosal hematomas may necessitate a period of from two to six months.



Serial x-ray photographs demonstrating mucosal hematoma formation and resolution.

9a — Normal sinus, July 6, 1943.

9b—Mucosal hematoma following altitude chamber flight, August 11, 1943.

9c—Resolution, January 25, 1944.

Complications. If the barotraumatized tissues do not become infected, recovery is usually rapid and unspectacular. Should the mucous membranes become infected, as rarely occurs, a severe purulent sinusitis may result. Hemorrhage into the cavity is not rare. As the hemorrhage is space-filling, it is a mechanism by which the inside and outside pressures are equalized, thereby releasing the flap valve and usually alleviating the symptoms other than epistaxis. Mucosal and submucosal hemorrhages are absorbed after a time.

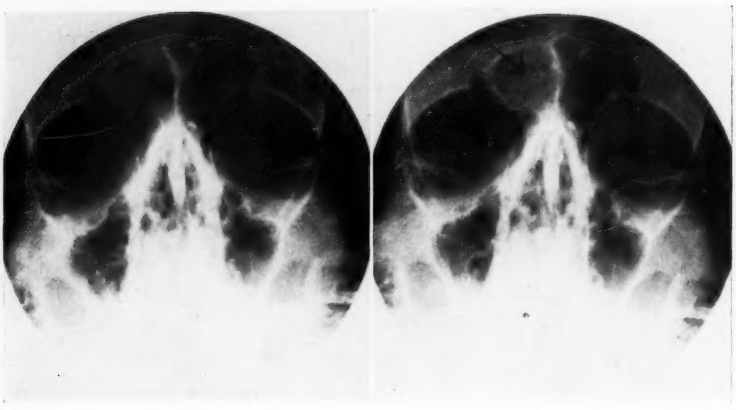
Treatment. From the standpoint of prophylaxis, those who have upper respiratory infections, nasal allergy, or nasal obstruction from anatomical deformity or polypi should not undergo great barometric pressure change until it is known that adequate nasal and sinal ventilation exists. This, of course, is difficult to ascertain exactly. However, in most cases freedom from obvious obstruction or pathology can be accepted as presumptive evidence. To err on the safe side, the Altitude Training Program has adopted a test flight to an altitude of 5,000 feet, which serves as an elimination procedure for those with either blocked sinuses or ears.

In developed cases, the otorhinolaryngologist is called upon to use his best judgment for establishing ventilation. Non-obstructive aerosinusitis usually responds to the simplest forms of treatment. Shrinkage of the offending structures will usually allow normal ciliary activity to evacuate a sinus of extraneous materials which may have been pressed into it.

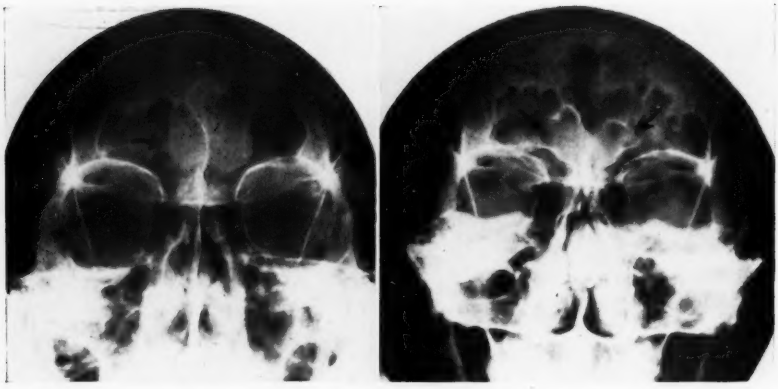
Obstructive aerosinusitis presents a somewhat different problem. Here the primary objective is to equalize the pressures inside and outside the cavity. A return to the altitude at which the block formed, then emptying the nose of secretion, followed by shrinkage of the offending tissues and slow return to earth would seem the ideal procedure. However, before attempting such a measure, one should know of the underlying pathology before subjecting the patient to further barotrauma.

Any procedure which will increase fluid production inside the sinus cavity will aid pressure equalization. Heat of some form thus seems indicated. At times it is necessary to mechanically move a turbinate or a polyp to break up the block. Usually, however, shrinkage by any of the usual vasoconstrictor agents will suffice.

Following recovery from aerosinusitis, the nasal passages should be carefully inspected and any obstruction removed before further



A. B.



C. D.

Fig. 10.—Random roentgenographic studies illustrating mucosal hematomas. *a* demonstrates aerosinusitis with hematoma. *b* same case, showing half resolution two months later.

exposure to altitudinal change. In a few instances, a submucous resection may be necessary.

In conclusion, it must be pointed out that the pressurization of aircraft should offer the best method for the prevention of any of the body's reactions to altitude or to barometric pressure change. Thus, as is often the case in aviation medicine, the ultimate solution to the Flight Surgeon's problem must, to a great degree, be made the responsibility of the aeronautical designer and engineer.

SCHOOL OF AVIATION MEDICINE.

REFERENCES

1. Bronk, D. W.: The Altitude Training Program During 1943, U. S. Army Air Forces Aviation Physiologists Bulletin, No. 3, January, 1944.

2. Bert, Paul: La Pression Barometrique, 1878. Translated by M. A. Hitchcock, and F. A .Hitchcock, Columbus, Ohio, College Book Company, 1943.

3. Marchoux, E., and Nepper: Compt. ren Soc. de biol. 82:668-673, 1919.

4. Behnke, A. E., and Willmon, T. L.: Physiological Effects of High Altitude, U. S. Naval Med. Bull. 39:163-178, 1941.

5. Brouwer, J. E.: Des limites aérophysiologiques imposées au pilote de l'avion de chasse, Arch. med. Belges (Bull. Internat.) 88:307-317, 1935.

6. Pastore, P. N.: Hearing Among Experienced Aviators, Proc. Staff Meet., Mayo Clin. 16:214-217, 1941.

7. Von Diringshofen, H.: Medical Guide for Flying Personnel, Tr. by V. E. Henderson, Toronto, Univ. of Toronto Press, 1940, p. 13.

8. Hermann, A.: Laceration of the Mucous Membrane and Hematoma of the Nasal Sinuses in Aviators, Ztschr. f. Hals-, Nasen- u. Ohrenh. 37:103, 1940.

9. Hermann, A.: Picture of Mucosal Detachment and Submucosal Hematoma of Nasal Sinuses due to Dive Flying, Ztschr. f. Hals-, Nasen- u. Ohrenh. 48:87-104, 1941.

10. Salem, W.: Aerosinusitis, Hospital, Rio de Janeiro 22:247-251, 1942.

11. Campbell, P. A.: Aerosinusitis—Cause, Course and Treatment, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 53:291-301, 1944.

12. Campbell, P. A.: Aerosinusitis, Arch. Otolaryng. 35:107-114, 1942.

13. Wright, R. W., and Boyd, H. E.: Aerosinusitis. Unpublished article.

VI

LOCAL INFILTRATION OF PENICILLIN SOLUTIONS IN PERITONSILLAR AND PHARYNGOMAXILLARY SPACES

MAJOR A. J. CONE, M.C., U. S. ARMY

ELLINGTON FIELD, TEXAS

The complications of acute infections of the lymphoid tissue of the naso- and oropharynx include: local areas of infection, as quinsy, retropharyngeal abscess, and pharyngomaxillary or parapharyngeal abscess; acute and chronic involvements of the ears, the paranasal sinuses and the cervical glands; systemic complications, as nephritis and rheumatic fever; bloodstream infections with multiple unpredictable sequelae (postanginal pyemia).

Removal of tonsils, drainage of peritonsillar abscesses and control of patients in whom postanginal sepsis is a threat, are all difficulties of particular significance in Air Corps personnel. Interruption of training programs and disruption of combat crews become necessary when vulnerable personnel must be grounded to protect them from altitude, fatigue, and extreme temperature changes. Accepted clinical concepts require an interval of three to six weeks following an acute attack of tonsillitis and/or quinsy before surgical removal of the tonsils is considered advisable. Further delay increases the risk of more serious recurrences with the added possibility of complications, such as rheumatic fever and nephritis. Baum[1] has advised tonsillectomy as the treatment of choice in what he described as second-stage quinsy:

> " . . . where we actually have pus lying between the tonsil and the superior constrictor muscle. Old scars may so distort normal relations that the pus migrates in various directions. In such cases it may follow the aponeurosis still further upward to a point in the nasopharynx, or burrow through the aponeurosis and constrictor into the neck; extension downward into the mediastinum having been reported in a few rare instances. . . . It is often necessary to dissect so far and so deep to reach the pus that the additional surgery incident to complete tonsillectomy adds nothing to the risk, and very little to the extent of the operation."

Baum[2] has also advised surgical removal of tonsils during the course of acute cervical adenitis. Tonsillectomy has never become an accepted measure when acute local conditions exist; however, the

advent of chemotherapy (sulfonamides and penicillin) permits earlier surgical interference.

The first report of postanginal sepsis in the English literature was made by Long[3] in 1912. His patient, a 28-year-old male, developed streptococcus thrombi in the internal jugular vein and cavernous sinus causing paralysis of the orbital muscles. The onset followed the drainage of a peritonsillar abscess. Goodman,[4] in 1917, added a case of primary jugular thrombosis. Mosher,[5] in 1918, stated, "When a peritonsillar abscess is complicated by pyemia, the pus which has accumulated in the pharyngomaxillary fossa through which the carotid sheath runs has infected the internal jugular vein and started, or produced a thrombus in it." Waldapfel[6] describes posttonsillar pyemia:

"Usually, though not invariably, a few days after the beginning of an attack of tonsillitis, a chill occurs. There is a slight tenderness over the region of the sternocleidomastoid. The patient looks septic. A pleuritis often occurs. There is usually fatal termination. Autopsy shows in many cases a thrombosis of the internal jugular vein on the affected side. Practically always a parapharyngeal abscess is found . . . . abscesses are found in the immediate neighborhood of the tonsil, thrombosis of the veins much more seldom, venous thrombosis without abscess formation almost never. Both thrombosis and bacterial invasion originate from the abscess. In the great majority of cases of posttonsillar pyemia, abscess formation occurs and invariably with the same location, viz.; in the anterior part of the parapharyngeal space, often multiple, connected only by the thrombosed parts of the veins."

Reuben,[7] in 1931, stated,

"In a number of cases the mere enucleation of tonsils in the course of sepsis caused a subsidence of temperature and complete recovery. . . . We believe that the local condition and infection should be eradicated first. If the symptoms persist, then it would seem that ligation of the internal jugular vein should be carried out."

Hall,[8] in 1939, in a report of septis following pharyngeal infection, stated:

"To control the primary fossa we must afford surgical drainage and/or institute chemotherapy. . . . The primary requisites of treatment are: (1) to control the primary focus; (2) to cut off the primary focus from the general circulation; (3) to combat metastatic foci."

Hill[9] has presented a most concise discussion. He states:

"Deep infection of the neck generally means pus in the pharyngomaxillary fossa. This is bounded internally by the superior constrictor muscle of the pharynx with the tonsil externally by the internal pterygoid muscle and parotid gland, and posteriorly by the prevertebral muscles and fascia. The carotid sheath with the great vessels of the neck is found in the posterior part and for practical purposes continues the fossa through the neck and into the chest. Infection is most often

secondary to infection in or about the tonsil, but may come from the parotid gland, from the floor of the mouth, from the petrous portion of the temporal bone, or from the retropharyngeal fossa. It may become infected from the needle in the administration of a local anesthetic agent preparatory to tonsillectomy, or in the performance of mandibular block."

In an attempt to evaluate the reactions and results of peritonsillar and parapharyngeal injections of penicillin, I have used local deep infiltration of penicillin in 20 tonsillectomies, and on six patients with quinsy (two of whom appeared to have a parapharyngeal abscess). Crowe[10] states:

"The local use of these substances to aid in the cure of sinus, ear, and pharynx infections has been ignored, with the exception of sulfonamide powders. . . . A second essential is to administer the antibacterial agent in such a way that it comes into actual contact with the infecting bacteria in effective concentration and for an adequate period of time. Both the concentration and the time factor are important."

Keefer,[11] et al, reporting on the mode of administration of penicillin, include the local injection into the pleura, pericardium and joints and into the subarachnoid space. They state: "It has not been possible to sterilize empyema cavities by the intramuscular or intravenous route."

In my personal experience, which is limited, intravenous, intramuscular and subcutaneous injections of penicillin have been of great value in acute infections of the ear, nose and throat. Local infiltration about the tonsil assumes a special position, largely because of the time element in relation to necessary surgery, and because the area of involvement is readily approachable by a simple injection. A higher local concentration of penicillin is obtained by local infiltration than is possible by other routes. Protection allowing the use of local anesthesia is provided.

REPORT OF CASES

CASE 1. A 28-year-old, white male entered the hospital with a history of sore throat, headache, fever and chills of one day's duration. His temperature on the day of admission was 104° F.; pulse 100. He was treated by local insufflation of sulfonamide powder and hot sodium chloride irrigations. On the third day after admission, marked peritonsillar involvement developed. The white blood count was 19,850 with 20 stabs, 67 segmented cells, and 1 juvenile. The throat culture showed general bacteria; 10,000 units of penicillin in 2 cc. of physiological sodium chloride solution were injected into the right peritonsillar region and parapharyngeal space. The patient com-

plained of sharp pain referred to the right occiput and the right parietal area. This subsided after two or three minutes. In the course of the next two hours he felt much better and was able to swallow without discomfort. There was no increase in the peritonsillar swelling. The next day 10,000 units of penicillin in 2 cc. of physiological sodium chloride solution, combined with 1 cc. of 1% novocain were injected into the parapharyngeal region. No pus was obtained by aspiration. The edema and redness in the right side of his throat cleared up.

The tonsils were removed by dissection and snare ten days after the first injection of penicillin, and one week later his temperature had returned to normal. At the time of operation, 5,000 units of penicillin were used as a deep retrotonsillar injection after the routine novocain infiltration. His postoperative course was uneventful and he was discharged to duty five days later.

CASE 2. A 20-year-old, white male entered the hospital with a history of having had one severe attack of tonsillitis each year. Four days before admission he had developed a severe sore throat. The examination showed marked peritonsillar swelling and redness of the right tonsil; his temperature was 99.6° F.; pulse, 120; white blood count, 11,600; segmented, 88; stabs 6; juveniles, 2. Ten thousand units of penicillin in 2 cc. of physiological sodium chloride solution, combined with 1 cc. of 1% novocain, were injected deep into the peritonsillar area in the involved side. No pus was obtained on aspiration. The following day the throat showed marked improvement; 10,000 units of penicillin in 2 cc. of physiological sodium chloride solution combined with 1 cc. of 1% novocain were injected. The pulse dropped to normal. There was very marked subjective and clinical improvement. The right peritonsillar infiltration cleared completely, but the patient developed peritonsillar infiltration of the opposite tonsil. On two successive days he received 10,000 units of penicillin in 2 cc. of physiological sodium chloride solution combined with 1 cc. of 1% novocain as a deep injection. All soreness cleared up, but the patient developed a localized fluctuant swelling at the upper pole of the left tonsil. One cc. of novocain and 2,500 units of penicillin were injected superficially. Pus was obtained on aspiration. The abscess was incised and drained without discomfort. The culture showed many organisms; streptococci predominated. Recovery was uneventful.

CASE 3. A 28-year-old, white male entered the hospital complaining of violent pain, difficulty in swallowing, and weakness. Six

weeks previously he had a mild sore throat; five days previously an acute sore throat. On examination there was marked bulging lateral to the right tonsil. Ten thousand units of penicillin in 2 cc. of physiological sodium chloride solution and 1 cc. of 1% novocain were injected deep into the parapharyngeal region, at which point pus was obtained on aspiration. The deep abscess was immediately incised, and about 5 cc. of pus escaped. There was immediate improvement in subjective complaints and of the trismus. Cultures of the pus showed nonhemolytic streptococci. Because of the severity of this patient's infection, he was given penicillin subcutaneously, 15,000 units every three hours. This was continued for ten days, at which time a tonsillectomy was done. As a prophylactic measure, 7,500 units of penicillin in 1.5 cc. of physiological sodium chloride solution were used as a deep peritonsillar injection after the routine 1% novocain infiltration just prior to tonsillectomy. The postoperative course was uneventful; healing of the fossae was unusually rapid. This patient undoubtedly had a parapharyngeal abscess.

CASE 4. A 37-year-old, colored male had been hospitalized for five days for treatment for sore throat. Vincent's infection was present on the first hospital admission. Two days after discharge from the hospital he began to have marked soreness on the right side of his throat. He was readmitted. A throat culture showed nonhemolytic streptococci. There was marked peritonsillar swelling. Five thousand units of penicillin in 1 cc. of physiological sodium chloride solution and 1 cc. of 1% novocain were injected deep in the peritonsillar area. Pus was obtained on aspiration from the parapharyngeal region. Incision was made through dense infiltrated tissue, and a very deep parapharyngeal abscess drained. The culture of pus from the abscess showed nonhemolytic streptococci; the urinalysis showed 2+ albumen, occasional red blood cells and occasional fine granular casts. The albumen, red blood cells, and casts cleared up after one week.

Tonsillectomy was performed two weeks after the drainage of the parapharyngeal abscess. A prophylactic injection of 7,500 units of penicillin in 1.5 cc. of physiological sodium chloride solution was given after routine novocain infiltration was used just prior to the tonsillectomy. There was no postoperative fever and no bleeding; healing was rapid.

Comment. Local anesthesia is unnecessary for the incision of most peritonsillar abscesses. In Cases 3 and 4 there was undoubtedly a parapharyngeal abscess. Adequate drainage would have been most difficult to obtain because of the marked trismus and dense infiltration

if local anesthesia had not been used. Without aspiration further time for localization would have been necessary.

No patient receiving peritonsillar or parapharyngeal injections of penicillin, alone or combined with novocain, showed any untoward reaction. The abscess cases were improved, two cases clearing without surgical drainage. Surgical drainage was facilitated in the two patients with parapharyngeal abscess.

Penicillin as a prophylactic local infiltration just prior to tonsillectomy was used in 21 patients during a three-month period. During the same period 28 routine local tonsillectomies were done. All patients were observed for reactions. Solutions were made in physiological sodium chloride solution and contained 5,000 units per cc. From 1 to 2 cc. were used as a deep peritonsillar injection just prior to tonsillectomy after the routine infiltration with novocain. In a few cases the penicillin was mixed with 10 cc. of 1% novocain. No instance of reaction, i.e., edema or hives, occurred. From a clinical standpoint healing was facilitated.

CONCLUSIONS

1. Penicillin in physiological sodium chloride solution in dosages of 5,000 to 10,000 units is well tolerated when used as a peritonsillar infiltration.

2. Penicillin combined with novocain has proved valuable when surgical drainage of peritonsillar or parapharyngeal abscess has become necessary.

3. Aspiration in cases of parapharyngeal infection permits earlier localization and drainage of collections of pus.

STATION HOSPITAL.

REFERENCES

1. Baum, H. L.: Radical Cure of Peritonsillar Abscess, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 35:429-437 (June) 1926.

2. Baum, H. L.: Tonsillectomy in Treatment of Acute Cervical Adenitis in Children, Tr. Sect. Laryng., Otol. and Rhin., A. M. A. 1930, pp. 97-120.

3. Long, J. W.: Excision of Internal Jugular Vein for Streptococcus Thrombin of Vein and Cavernous Sinus Causing Paralysis of Orbital Muscles, Surg. Gyn. & Obst. 14:86, 1912.

4. Goodman, Charles: Primary Jugular Thrombosis Due to Tonsil Infection, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 26:527, 1917.

5. Mosher, H. P.: Thrombosis of the Internal Jugular Vein with Pyemia as a Complication of Retropharyngeal Abscess, Laryngoscope 29:648, 1919.

6. Waldapfel, Richard: Post Tonsillitis Pyemia, Tr. Amer. Acad. Ophth. & Otolayryng. 1928, pp. 291-296.

7. Reuben, M. S.: Post Anginal Sepsis (Sepsis of Oro-Naso-Pharyngeal Origin), Arch. Dis. Child. 6:115-128 (April) 1931.

8. Hall, Colby: Sepsis Following Pharyngeal Infections, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 48:905-925, 1939.

9. Hill, F. T.: Ophthalmology and Otolaryngology; Military Surgical Manuals, National Research Council, Philadelphia, W. B. Saunders Company, 1942, pp. 289-290.

10. Crowe, S. J.: The Local Use of Sulfadiazine Solution, Radon, Tyrothricin and Penicillin in Otolaryngology, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 53:227-241 (June) 1944.

11. Keefer, C. S., et al.: Penicillin in the Treatment of Infections, J. A. M. A. 122:1217-1224 (Aug. 28) 1943.

VII

AN ADAPTATION OF THE DISPLACEMENT PRINCIPLE FOR THE INTRODUCTION OF GASES INTO THE SINUSES

ARTHUR W. PROETZ, M.D.

ST. LOUIS, MO.

The Displacement Method was devised for the purpose of introducing fluids into the nasal sinuses with the least possible traumatization of their ostia.

With the advent of effective vasoconstrictors in gaseous form, a method seems desirable for introducing these also into the sinuses. Inhalers, even those of today which are vastly superior to the earlier ones, have one shortcoming in common with the atomizer, namely, that the drug does not penetrate the ostia, especially those blocked with secretions.

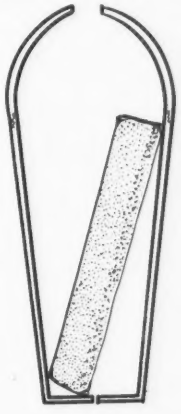


Fig. 1.

A simple means is here presented of applying the Displacement principle to gases.

The appliance consists simply of a bomb-shaped container, similar to those now used for most inhalers and carrying the familiar cotton cylinder saturated with the volatile drug. A *small* hole is drilled in each end of the container.

Using the same pumps, the same pressure (minus 180 mm. Hg.) and the same olive tip as for displacement with fluids, intermittent suction is applied at one nostril while the patient keeps the mouth open and the velum palati in the "k" position. Naturally no fluid is introduced.

Now, instead of closing the opposite nostril with the finger, the perforated olive tip of the container is inserted snugly into it. The negative pressure draws the fumes into the nose from the container and at the same time evacuates part of the air from the sinuses. With its release small quantities of vapor-laden air are carried into the

Fig. 2.

sinuses. The pressure is applied a number of times as in displacement with fluids.

It is absolutely essential that the small hole at the distal end of the container be not larger (and it may be smaller) than 1/64 inch in diameter—in other words a pin hole. If this hole is any larger, then it becomes impossible to attain the necessary negative pressure in the nose to exhaust air from the sinuses.

The hole in the proximal olive-tipped end which is inserted in the nostril may be of any convenient size but should be somewhat larger than 1/64 inch. Experiments with a model show that if this hole is too small, the vapor enters the nasal chamber in an undesirable jet rather than in an evenly distributed stream.

It is axiomatic that the cleaner the nose the more effective the penetration will be. Therefore, in most cases it is advisable not only to shrink the nose but to cleanse the meatuses as far as possible with a thin suction tube.

It may, at first glance, seem immaterial whether the alterations in negative pressure be made from the side of the suction tip or through the container. However, the latter practice is not effective, since the release of pressure through the pin hole is too gradual to allow complete equalization.

One should exercise some caution in the choice of a volatile constrictor which is liberally used and which may be retained for a time in the sinuses. Our own experiments have been made with a preparation of 2-amino-heptane which has been shown to exhibit a minimal systemic effect.[1, 2]

While gases are less effective than fluids, they have this advantage: that they do not spread the infection and may be used in the acute stages of the attack.

This maneuver is somewhat reminiscent of an experiment described by Coffin to the American Laryngological Association in 1922.[3] In this paper Coffin describes a tube having two valves which permitted the alternate exhaustion of air in the introduction of vapor from a nebulizer. History in repeating itself usually adds a few embellishments. In this case one might regard as an embellishment the volatile constrictors which did not exist in Coffin's day, were it not that here they constitute the cardinal factor in effectiveness.

It will be unnecessary to add that since gravity does not play a part, the usual Displacement position is dispensed with and the patient kept in the erect position.

1010 BEAUMONT BUILDING.

REFERENCES

1. Proetz, A. W.: Certain Aliphatic Compounds as Nasal Vasoconstictors, Arch. Otolaryng. 37:15-22 (Jan.) 1943.

2. Chen, K. K., and Swanson, E. E.: Cit. Ref. 1.

3. Coffin, R. A.: Experiments with Atmospheric Pressure in the Maxillary Sinus, Tr. Am. Laryng. Assn., 1922, pp. 291-297.

VIII

CILIA AND PENICILLIN

A LABORATORY NOTE

ARTHUR W. PROETZ, M.D.

ST. LOUIS, MO.

Preliminary to an investigation of the effects of penicillin on sinus infections, strips of ciliated respiratory epithelium were subjected to the drug in various concentrations with the following results:

Periods for which the ciliary beat persisted during immersion were

250 units per cc.	—	25	hr.	50	min.	to 27 hr. 45 min.
500 units per cc.	—	15	hr.	25	min.	to 19 hr. 35 min.
5000 units per cc.	—	3	hr.	20	min.	to 6 hr. 25 min.
All controls	—	27	hr.	35	min.	to 28 hr. 20 min.

The method of preparing the specimen and applying the solution differed somewhat from those employed in previous studies of other drugs. It was considered possible that penicillin solutions remaining in sinuses for protracted periods might injure the membrane; therefore specimens were immersed in .5 cc. of solution and permitted to remain there instead of washing or replenishing with fresh solution from time to time.

A rabbit was killed and the trachea was isolated, clamped at both ends and severed beyond the clamps to protect the lining from blood, air and foreign matter. The trachea was transferred at once to a covered Petri dish containing a small amount of physiological sodium chloride solution. It was cut up into rectangles .5 cm. long and one third the circumference of the trachea in width, which yielded about thirty specimens. Leaving the mucosa attached to the cartilages facilitates handling and the slight curvature of the surface produces highlights necessary for detecting motion.

The specimens were placed on Maximow slides, immersed in .5 cc. of the test solutions and kept in covered Petri dishes. For observation they were withdrawn from the pool of solution and the excess fluid was removed with a fine capillary pipette. They were studied quickly and returned to the solution without delay to prevent drying.

Tissues were kept at approximately 30° C.

Experience has shown that this simple method, using tracheal mucosa, is as informative as the more cumbersome ones using sinus linings and living animals. In all cases the effects of drugs have been the same for sinus and tracheal mucosa of animals in situ and in vitro, and for human specimens removed at operation.

The tests with penicillin indicate that solutions in the strength recommended for local application (250 units per cc. isotonic NaCl solution) has no appreciable adverse effect on respiratory ciliated epithelium. The ciliary beat ceased only when the epithelium disintegrated, which it did after some 27 hours. This occurred at approximately the same time in the controls.

In solutions of 500 units per cc. the active period was somewhat shortened (average 18 hours) and the beat was less vigorous throughout.

In solutions of 5000 units per cc. the beat was reduced to approximately half-speed within a few minutes of immersion and continued so, without further retardation, until it stopped completely in 3½ to 6½ hours, without any sign of surface disintegration. While this concentration has not been recommended for topical application it is useful to know that the stronger solutions are less well tolerated than the weaker ones.

Two specimens immersed in solution of 12.5 units per cc. differed in no way from the controls.

The material used was the sodium salt of penicillin, supplied in ampoules of 100,000 units and was obtained from the druggist's stock.

CONCLUSIONS

Solutions of penicillin (sodium salt) in the concentrations recommended for topical application have no damaging effect on the cilia or the epithelium of the respiratory mucosa over periods useful in clinical practice.

Solutions of higher concentration tend to impede ciliary action in various degrees.

1010 BEAUMONT BUILDING.

IX

BRIEF LABYRINTHINE PHYSIOLOGY FOR RESIDENTS AS PART OF BÁRÁNY INSTRUCTION AT THE MASSACHUSETTS EYE AND EAR INFIRMARY

FRANCIS L. WEILLE, M.D.

BOSTON MASS.

The anatomical facts relating to the labyrinth which are of physiological importance should be evaluated not only for hearing, caloric and rotation tests, but also for labyrinthine complications of the middle ear and mastoid infections and for the fenestration operation.

The labyrinthine capsule is said to be the hardest bone in the body. It is very much larger than is ordinarily realized, forming as it does an oblique wall-like mass in the petrous base, separating the middle ear and mastoid from the more medial portion of the petrous pyramid. Usually with a fringe of perilabyrinthine cells, it comprises the "base" of the petrous pyramid. The cochlea and vestibule lie medial to the middle ear while the bony mass enclosing the semicircular canals lies medial to the mastoid. Of the three canals, the horizontal is the shortest (12 to 15 mm.). With the head erect this canal is not really in the horizontal plane but slants upward about 30°; hence, to have this canal parallel with the floor in rotation tests, the head is bent forward approximately 30°. The longest canal is the superior, which is placed exactly at right angles to the posterior surface of the petrous pyramid in a vertical plane. It makes a mound on the anterior superior surface of the petrous pyramid called the arcuate eminence. This canal can be made to approximate the horizontal plane by bending the head straight forward 120°. So can the posterior canal, which lies in a vertical plane exactly parallel with the posterior surface of the petrous pyramid.

Each canal has an ampulla at its anterior end about twice as wide as the arch of the canal. The two vertical canals join posteriorly so that there are only five openings of the three canals in the bony vestibule—the three ampullae, the posterior end of the horizontal canal, and the common posterior end of the vertical canals.

The bony vestibule measures about 5 by 5 by 3 mm. and joins the bony cochlea with the canals. The cochlea, simulating a snail-shell of two and a half turns, would measure about 31 mm. in length if uncoiled. Actually, it measures 6 mm. from the oval window to the apex anteriorly (called the cupola).

The bony labyrinth contains spinal fluid, called perilymph, in which lies the membranous labyrinth consisting of the cochlear duct which contains the organ of Corti, the ductus reuniens, the saccule, the utriculosaccular duct, the ductus and saccus endolymphaticus and the utricle into which enter the membranous semicircular canals. The membranous labyrinth contains endolymph which is said to be secreted from vessels (stria vascularis) in the spiral ligament of the cochlear duct, and excreted by absorption in the saccus endolymphaticus. The aquaeductus cochleae is a conduit for spinal fluid from the region of the subarachnoid space of the jugular fossa to the first turn of the cochlea; the aquaeductus vestibuli contains the ductus and saccus endolymphaticus and ends blindly between layers of the dura of the posterior fossa. The saccus is said by Braun[1] to project beyond the aqueduct into layers of the dura at the medial aspect of the vertical limb of the lateral sinus.

The vestibular ganglion lies in the fundus of the internal auditory meatus. This ganglion has dendrites supplying the membranous canals, the saccule, and the utricle. The cochlear, or spiral, ganglion lies beyond the internal auditory meatus in the modiolus of the bony cochlea, and has dendrites supplying the hair cells of the organ of Corti. The axons of these ganglia comprise the trunk of the eighth nerve.

Infections within the labyrinth may spread intracranially through the internal auditory meatus, or the aqueducts. Changes in spinal fluid pressure may be transmitted through the aquaeductus cochlea to the perilymph. The tip of the saccus endolymphaticus is so situated anatomically as to be vulnerable both to changes in spinal fluid pressure and venous pressure. (Venous and spinal fluid pressure, of course, must vary together physiologically). Pressure changes in the membranous labyrinth are demonstrable pathologically in Ménière's syndrome.

The most important anatomical structures in relation to the Bárány tests are the membranous semicircular canals and the utricle. Obviously cochlear function must always be examined. The recognized pathways of the vestibular nerve must be known to the examiner in order to understand, for example, induced nystagmus and

falling. Before discussing such pathways, one must understand the basic physiology of the inner ear.

The cochlea functions as a hearing receptor, while the utricle and membranous semicircular canals are organs of equilibrium.

The utricle is believed to function in relation to "static" equilibrium: that is, as an important factor in "tuning" the body at rest to its position in space. It functions similarly (Tait[2]) in very slow tilting motions of the head insufficient for stimulation of the semicircular canals (the "kinetic" labyrinth); and in linear acceleration in any direction. Its prime stimulus is gravity. Clinically it produces compensatory eye positions, head righting reflexes, and alterations in muscle tonus of the entire body, but only the last of these is useful in clinical work or in the Bárány test.

The function of the saccule is unknown.

The semicircular canals represent the "kinetic" labyrinth, in that their function comes into action physiologically only with rotation of the head (angular acceleration) regardless of direction. Both labyrinths function together in this manner, and never unilaterally except in the caloric test. This reciprocal function of the two labyrinths has clinical importance: for example, with sudden destruction of one labyrinth the resulting clinical picture is due to the overfunctioning of the remaining labyrinth. The prime stimulus of the semicircular canals is inertia.

The relation between the cochlea and sound stimuli is as follows:

Air-borne sound waves have physical energy which may be converted into other forms of energy such as light or electricity and so measured. With physiological modifications this fact is the basis of much experimental data collected from investigations of the ear. By "feeding" in sound waves and observing resulting electrical disturbances with appropriate apparatus, it is possible, for example, to photograph the electrical cochlear potentials called "cochlear microphonics" when they are converted (by a cathode ray oscillograph) into light energy. Such information affords highly useful facts regarding the mechanism of cochlear function.

The principle of conversion of energy from electricity into sound is utilized in the audiometer tests. With the audiometer it is possible to maintain any desired frequency but to vary the amplitude of vibration and thereby vary the intensity of the testing sound. The energy of the test sound is measured in decibels. The decibel is ap-

proximately equivalent to a root—mean—square pressure of 0.002 dyne per square centimeter. A dyne is equivalent to the gravity force exerted by a milligram weight. The extreme delicacy of hearing function is not commonly realized. It has been possible electrically to demonstrate[3] that where the ear is most sensitive an amplitude of vibration of the ear drum less than the diameter of a hydrogen molecule produces a sound stimulus in the basilar membrane. A displacement of the basilar membrane equal to less than one per cent of the diameter of a hydrogen molecule will at 3,000 cycles produce the sensation of hearing.

The middle ear permits the conversion of sound waves in air to fluid pressure disturbances in the cochlea. Such external stimuli are "exteroceptive."

"Proprioceptive" stimuli originate within the body itself—for example in the utricle, the semicircular canals, and in muscles, tendons and joints.

The inner ear, then, normally deals with both "exteroceptive" sensation in the cochlea and "proprioceptive" sensation in the utricle and canals. These facts are of some importance in understanding the physiology of the inner ear, especially in relation to nerve pathways from the ear to the brain and spinal cord.

Contrary to current opinion, a simple knowledge of these pathways can be very useful, and is easily acquired. Thus, the axon from the ganglion cell of the vestibular ganglion proceeds, as the vestibular nerve, to the vestibular nucleus in the floor of the fourth ventricle. An easy way to keep in mind the position of this nucleus is to regard the diamond-shaped fourth ventricle as a baseball diamond with the vestibular nuclei of the right and left ears lying laterally at first and third base. It is well-known that these nuclei are connected with the ventral horn cells (i.e. motor cells of the spinal nerves) of the spinal cord through a "ground bundle" called the vestibulospinal tract. It will be recalled that ground bundles of connecting fibers join various levels of motor cells of the spinal cord with each other as the simplest possible pathways. The vestibulospinal tract is merely a "ground bundle" which completes a reflex arc from the vestibular nucleus to any motor cell of the spinal cord. This arc permits the falling reaction in vestibular tests to be about as reliable as nystagmus responses in Bárány tests; over both responses—falling and nystagmus—the patient has no subjective control.

The connection of the vestibular nuclei with other cranial nerves through the medial longitudinal bundle can also be understood by

regarding the fourth ventricle as a baseball diamond. An imaginary line drawn between first and third bases (as described above, corresponding to the right and left vestibular nuclei) reaches of course to "pitcher's box"—the position of the sixth nerve nucleus. This line then turns at a right angle to reach second base and center field (positions of the motor nuclei of the fourth and third nerves respectively, ventral to the aqueduct of Sylvius at the inferior and superior corpora quadrigemina levels of the midbrain). These fibers are, again, merely "ground bundles" which connect the sixth, fourth, and third cranial nerve brain stem nuclei with the vestibular nuclei. This simple pathway affords a direct connection between the vestibular nucleus and the motor nerves to the extra-ocular muscles, and thereby permits nystagmus to occur on vestibular stimulation.

Incidentally, the same "ground bundle" (medial longitudinal bundle) descends also from the "pitcher's box" to "home plate" and "batter-up" positions to connect with the twelfth and eleventh cranial nerve nuclei. Through connections between the vestibular nuclei and the cervical portion of the spinal accessory nerve, torsion of the neck can occur from vestibular stimulation.

The dorsal motor nucleus of the vagus nerve lies directly beside the vestibular nucleus on each side. In many patients who undergo maximal vestibular stimulation, nausea results because of the anatomical proximity of these nuclei.

It is known that the vestibular nuclei are connected to the vermis of the cerebellum, and to the cerebellar nuclei. While the major function of the cerebellum seems to be the integration of proprioceptive impulses for adequate striated muscle tone and synergistic muscular responses subservient to volition, a discussion of cerebellar physiology must, for the sake of brevity and simplicity, be omitted from this syllabus.

With the foregoing broad outline in mind, it is possible to understand more clearly details of labyrinthine physiology regarding hearing, nystagmus, falling, vertigo, past-pointing, equilibrium.

These will be discussed consecutively.

Hearing. The best treatise published to date on the physiology of hearing is that by Davis and Stevens[3] published in 1938. The second half of this book has been summarized by Fowler in Nelson's "Medicine of the Ear." A recent publication of importance is "The Response of a Single Auditory-Nerve Fiber to Acoustic Stimulation" by Galambos and Davis.[4]

The reason for discussing the physiology of hearing in relation to Bárány tests is self-evident. The cochlear duct, containing the organ of Corti, is directly continuous with the membranous structures of the vestibule and valuable information may be lost if all parts of the membranous labyrinth are not tested so far as possible; therefore, for a diagnosis of "dead labyrinth" to be made, there must be neither vestibular nor cochlear responses.

The energy of air-borne sound waves is converted into labyrinthine fluid pressure disturbances through the oval or round window or both. Inward displacement of perilymph at the oval window, therefore, must be followed by outward displacement of the round window, as the fluid itself of the labyrinth is not compressible. Hydrodynamic wave patterns* in the scala vestibuli and the scala tympani compress the cochlear duct at various levels depending upon the types of waves set up by the stimulating sounds. Experimental evidence[3] indicates that specific sounds cause responses in "place areas" of hair cells of the organ of Corti over specific portions of the basilar membrane. In turn, through hair cell stimulation, nerve fiber responses occur. Compression of hair cells by the fluid wave patterns probably causes a piezoelectric† effect resulting in the production of electrical potentials. Such potentials are the "aural-microphonics." The aural-microphonics have a latent or refractory period about one-tenth that of a nerve impulse. Thus, the refractory period of a nerve fiber is approximately 0.001 of a second or one m. sec.; that of the aural-microphonic is so very much smaller that it is relatively easy to distinguish electrically between the two.

It is very clarifying to the student's mind to understand three specific physiological problems in the hearing mechanism: (1) how a low tone is heard; (2) how a high tone is heard; (3) masking. The first and second factors will be discussed together.

These factors have been studied by means of the responses of a single auditory nerve fiber to acoustic stimulation and reported by Galambos and Davis.[4] It has been found that the refractory period of auditory nerve fibers is approximately 0.001 of a second. It is not possible, therefore, for any one fiber to carry more than 900 impulses per second. Nerve tissue rarely functions at such top speed in any

*Pascal's law: Pressure exerted anywhere on a confined liquid is transmitted undiminished to every portion of the interior of the container. In the cochlea this law aids place area stimuli of the basilar membrane because of the varying width of the basilar membrane.

†Quartz and certain other crystals become electrified when subjected to pressure or tension. This is called piezoelectricity.

part of the body. This is true of the auditory nerve also. The average impulses per second are about 300 to 400 ordinarily. This is true whether the tone which is being transmitted is a low one with only a few cycles per second or a high one with many cycles per second. When the intensity of the sound stimulus is fixed at a value just sufficient to cause a minimal response, only a very narrow band of sound frequencies excites a given auditory nerve fiber, according to Galambos and Davis, so that to each fiber may be assigned a characteristic frequency band in a particular minimal intensity.

For low tones this band lies within about plus or minus ten cycles per second and for high tones within about one hundred cycles per second. That is, each fiber "tunes in" sharply to a specific and narrow region of the sound spectrum. This finding is of supreme importance in establishing the mode of action of the cochlea in hearing.

The range of sound frequencies capable of exciting a fiber becomes more extensive as the intensity level is raised. That is, the sharp tuning noted at minimal intensity disappears as the intensity level is raised. At 90 db. above minimal intensity all frequencies between 250 c.p.s. and 2500 c.p.s. respond to a 2000 c.p.s. sound stimulation. That is, larger and larger areas of the basilar membrane respond to increasingly more intense tones. In this instance there are three octaves below and one-half octave above the characteristic frequency response.[4]

The rate of discharge of a single auditory fiber is determined by several factors, the most interesting of which is the frequency. The frequency of nerve discharge is not determined by the stimulus frequency per se but is the function of both the frequency and the intensity of the stimulus. A high tone merely stimulates specific fibers from a particular portion of the cochlea (first turn); and a low tone, fibers from another portion (third turn). The response to a very high tone causes no particular change in the number per second of nerve fiber responses that is different from the stimulus resulting from a low tone. Therefore, a 512 db. stimulus does not cause 512 nerve impulses per second, or 8192 db. 800-900 nerve impulses per second in 10 fibers.

Clinical masking is based on the principle of producing a refractory period in all fibers of the auditory nerve of the ear not being tested. That is, this nerve gives a "busy signal." In order to produce such an effect the masking tone must be such that it is not heard by the ear being tested. Air-borne sound fed into the ear through a well-built ear receiver requires more than 40 decibels intensity to

be heard through the skull by the opposite ear, at least in the low and middle tone ranges.

Hearing tests should be carried out in a soundproof room. For practical purposes, a noise-level of -25 db. is permissible. From time to time a noise-level meter should be used to check the sound level at various times of the day in a room constantly used by an examiner if his work is to be accurate.

Nystagmus. A fundamental function of the labyrinth is to aid in fixation of the field of vision. If, when one is shaving himself each morning, it is observed in the mirror how the eyes remain fixed on the skin being shaved as the head is moved, it should be remembered that the perfect coordination of this process depends upon the semicircular canals. From what has been said regarding the brain stem connections of the vestibular nerve, the nerve pathways of the mechanism can be easily recalled. Furthermore, if slight motions of the head cause relatively small compensatory movements of the extraocular muscles in order to fix the field of vision, it is obvious that severe stimulation of the ear, as by rotation in a chair or by pouring cold water into the ear, would result in infinitely greater eye responses. Such responses are jerking motions of the eyeballs and are called "nystagmus".

Nystagmus has two components: a slow motion in one direction with a quick jerk in the opposite direction. The quick jerk has arbitrarily been selected as denoting the "direction" of the nystagmus. Horizontal nystagmus is in the horizontal plane. Vertical nystagmus is in the sagittal plane. Rotary nystagmus is in the frontal plane. Oblique nystagmus is in the oblique direction to the right or left, up or down. Perverted nystagmus is the wrong type of nystagmus such as rotary instead of horizontal. Inverted nystagmus is in the opposite direction to which it is expected. Exaggerated nystagmus is large size excursions of the eyes or prolonged duration. Conjugate deviation is drawing both eyes to one side (slow component) with absence of the quick jerk (quick component). The amplitude of nystagmus refers to the size of the excursions of the eyes. Ophthalmic nystagmus is caused by pathological lesions of the maculae of the eyes so that constant motions of the eyes in irregular directions are necessary for clarity of vision. This last nystagmus is so easily recognized as never to be confused with aural nystagmus. First degree nystagmus is that seen only when the eyes are turned toward the quick component. Nystagmus present when the eyes look straight ahead is called second degree. Third degree nystagmus is observed with the eyes turned toward the slow component.

The fundamental laws of nystagmus now widely accepted as facts are as follows:

A. Flouren's Law: Injury to a canal causes nystagmus in the plane of the canal which is injured.

B. Destruction of one labyrinth in man still permits inducing nystagmus in either direction by turning, but the responses are not equal. After a time the responses, however, become equal.

C. Ewald's Laws:

1. The horizontal canal is maximally stimulated by movement of endolymph toward its ampulla; the vertical semicircular canals are maximally stimulated by a movement of endolymph away from the ampulla.

2. Maximal stimulation of any canal produces nystagmus to the same side; minimal stimulation to any canal produces nystagmus to the opposite side.
(The quick component of the nystagmus determines its direction in the above laws.)

D. The Hyndman-Bárány Laws. These have recently been reemphasized by Hyndman[5] who credits them to Bárány:

1. With rotation of a patient in a chair the plane of nystagmus is fixed to the plane of the floor and does not change with change in position of the head. Thus, rotation with the head placed obliquely produces oblique nystagmus. With the head turned to the side, vertical nystagmus results. With the head 30° forward, horizontal nystagmus occurs, and with the head 120° forward, rotary nystagmus is seen. Once the nystagmus has been induced, as rotary nystagmus after turning with the head 120° forward, changing the position of the head, as by sitting up, will not affect the nystagmus.

2. With the caloric test the plane of nystagmus is in the plane vertical to the floor. The nystagmus will change with change in the position of the head but always remains in the plane vertical to the floor.

It is true that Ewald actually produced motion of the labyrinthine fluid in his experiments. Our clinical methods of stimulation of the receptors of the semicircular canals are not established as being

due to actual currents in the endolymph produced by the rotation or caloric techniques. The effects of inertia especially in the endolymph of the utricle are important in effecting stimulation of the semicircular canals because of changes in pressure and tension.

Undric and Undritz[5] agree that the minimum rotation perceptible by the semicircular canals is 3° per second.

Falling. It has been pointed out that a pathway exists between the vestibular nucleus and the ventral horn cells of the entire spinal cord. It is, therefore, easy to understand how normal tonic reflexes from the labyrinth can be influenced by extreme stimulation of the ear as in the rotation or caloric tests to produce falling. Normally the falling is always toward the stimulated ear. It should be pointed out that when a patient is rotated with the head at 120° forward, thereby stimulating the four vertical canals, violent falling normally occurs when the patient sits up. This falling is to the right if the patient has been rotated to the right and to the left if the patient has been rotated to the left. Rotation with the head 30° forward to stimulate the horizontal canals never produces falling because the plane of the canals and the macula (otolith organ) of the utricles is parallel with the floor.

Labyrinthine reflexes causing striated muscle tonal responses originate in the utricle. Normally,[5] a motionless mammal has a maximal tonal response from the utricles of both ears when lying on the back; a minimal response from the left ear and a maximal response from the right ear when lying on the right side and vice versa, and a minimal response from both utricles if in the prone position. It is possible that falling in the Bárány tests is intimately related to disturbances of such normal tonal relationships.

Past-pointing. It has been pointed out in the nerve pathways described above that the medial longitudinal bundle connects the vestibular nucleus with the cranial portion of the eleventh nerve. In any event, through this bundle and through the vestibulo-spinal tract there is an intimate relationship between the vestibular nucleus and the motor origins of the eleventh nerve. It has been observed[5] that if past-pointing to the right, for example, has been induced after rotation to the right, rotation of the head to the right increases the past-pointing while turning the head to the midline lessens or abolishes such past-pointing. Such modification of past-pointing can be derived only from neck reflexes or the utricle.

Vertigo. While the fact of rotational vertigo is known to everyone the nervous pathway from the vestibular nucleus to the

sensory cortex is unknown anatomically. A possible pathway is the medial lemniscus.

Equilibrium. Equilibrium depends upon the following:

1. Exteroceptive sensation: vision and touch.
2. Proprioceptive sensation from muscles, tendons and joints; and from the vestibular portion of the inner ear.

Such afferent impulses are integrated and coordinated by the brain to produce effective motor responses resulting in the maintenance of equilibrium.

THE TECHNIQUE OF THE ROTATION AND CALORIC TESTS

First, do routine air and bone conduction audiograms and clinical hearing tests.

Second, determine whether or not the patient has spontaneous nystagmus by having him look straight forward while sitting in the chair in which he is to be tested. Then have him look to the right, to the left, upward and downward successively, and write down on the chart shown in Fig. 1 a zero for absent nystagmus, which is the normal.

Normally he should have no vertigo or past-pointing. Falling or staggering when attempting to walk 10 feet in a straight line with the eyes closed should be absent. Past-pointing is determined by having the patient with his eyes open point to the examiner's finger with the hand and wrist and fingers in the position of a signboard. Keeping the elbow extended and the eyes closed, the arm is raised so that the forefinger points toward the ceiling, and then returned to the original position pointing at the examiner's finger. This test is done for each arm, and the result recorded on the chart.

An inquiry is made as to whether or not the patient complains of vertigo at the time of the examination, and the response is written down.

A Romberg test is done in the usual manner. It is then repeated with the head turned to the right and again to the left.

The "overthrow" is done with the patient in the Romberg position, followed by bending the body laterally to one side, then the other.

The "spontaneous" tests are completed when the foregoing responses have been recorded.

A fistula test in chronic suppurative otitis media or in "effectus" otitis media may permit omission of further examination if it is positive, and if it is desired merely to be certain that the labyrinth is viable. The reason for the great importance of knowing whether or not the labyrinth in question is "alive" or "dead" is that grave decisions relating to the surgical technique for restoring the patient to health may be involved. For example, if a patient who has chronic suppurative otitis media with mastoiditis spontaneously develops a facial paralysis suddenly, a radical mastoidectomy would be done, but what further? It has been observed that the pathology causing such a facial paralysis usually lies in the labyrinthine capsule.[6] If such a labyrinth were "dead" it would be entered without risk of meningitis or other intracranial complications in the course of facial nerve surgery. If the labyrinth were "alive", breaking through the labyrinthine capsule would be a serious matter. In the former case, the labyrinth would probably be exenterated; in the latter, an effort would be made not to open it.

If, in the same case, the fistula test is negative, much importance attaches itself to (1) the hearing and (2) douching the external auditory canal with iced 70% alcohol; (3) and possibly to rotation tests described subsequently.

There is no disadvantage to pouring cold 70% alcohol into a draining ear. Therefore, 100 to 200 cc. of 70% alcohol are prepared in a sterile glass or enamel basin which itself is allowed to remain in a bath of crushed ice. The chilled alcohol is then slowly instilled into the external canal of the draining ear and the appearance of nystagmus noted as present or absent over a period of five minutes. If no reaction occurs—of any degree—and no hearing can be demonstrated with adequate masking of the other ear, the labyrinth tested is "dead". But if *any* response is present, the labyrinth is "alive."

If the patient's general condition allows it, rotation tests can be done for further confirmation.

The foregoing method applies to any chronic suppurative otitis media case where qualitative information only must be obtained.

Rotation tests are "physiological" in that both labyrinths are stimulated simultaneously. Both horizontal canals (not the vertical canals) are stimulated when the patient is rotated with the head 30° forward. All vertical canals (but not the horizontal canals) are excited when the head is placed 120° forward from the erect position and the patient rotated.

The patient is rotated 10 times to the right in 20 seconds, with a response normally of second degree nystagmus to the left for 16 to 24 seconds. He is turned with the eyes closed, and opens them when the turning has stopped. He is asked to state when he feels that the room has ceased to turn after the chair has stopped, and this is measured in seconds by means of a stopwatch, as is the nystagmus. This obviates the necessity of turning him 10 times to the right in 10 seconds to determine the length of vertigo response, which, with the faster turning is normally 16 to 26 seconds. With this modification of technique the normal vertigo response is 16 to 24 seconds—the same as for nystagmus. It is not usually necessary to measure past-pointing which is normally 10 to 12 inches to the right on rotation to the right 10 times in 10 seconds.

The same single test is repeated to the left with the direction of responses thereby reversed, but with the time rsponses normally the same. The results are recorded on the chart.

Rotation to the right stimulates the left horizontal canal maximally, and the right horizontal canal minimally; and vice versa with left rotation. See the text above relating to the laws of nystagmus.

Rotation to the right with the head 120° forward, the forehead resting upon the rectangular plate on the circular safety bar of the chair is then done.

The resulting nystagmus is rotary, to the left, and of 15 to 20 seconds' duration. Since the patient must sit up for the nystagmus to be observed by the examiner, the falling reaction to the right can be simultaneously measured (along with vertigo as described for the horizontal canal tests).

The test is repeated with rotation to the left. This reverses the direction but not the time of the responses. The results are recorded in the Bárány chart.

This test with the head 120° forward on turning to the right maximally stimulates the left vertical canals and minimally stimulates the right vertical canals; on turning to the left, vice versa.

"Maximal" and "minimal" stimulation are in a ratio of preponderance as 2 is to 1.

The caloric tests are "massive douching" and the Kobrac method. These tests can be done independently of the rotation tests or combined with any portion of the latter. The caloric tests stimulate

one ear at a time and may differentiate between the horizontal and the vertical canal responses. With any caloric technique there is a latent period between the time the irrigation starts and the occurrence of nystagmus.

The Kobrac technique is a "minimal stimulation" method. It requires the injection of small quantities of cold water into the external auditory canal.

The following are summaries of three modifications of the Kobrac test:

1. Mysel Test: Five cc. of ice water at 40° F. are used. The latent period is 15-30 seconds. The duration of the nystagmus is 30-60-75 seconds.

2. Lurie Test: Ten cc. of water at 35° F. (ice water) are injected into ear over a period of 45 seconds. The latent period is 15-30 seconds. The duration of the nystagmus is 30-60 seconds. If no response, wait 5 minutes and repeat with 20 cc. of water at 35° F.

   Use +20 glasses on the patient; look for second degree nystagmus.

3. Demetriades-Mayer Test:[7] Five cc. of water at 55° F. are injected slowly in the direction of the eustachian tube. The latent period is 15-30 seconds. The duration of nystagmus is 60-120 seconds. If there is no response after 3-4 minutes, tilt head backward 60° and turning toward the syringed side, repeat with 10 cc. of water at 55° F. In this test look for first degree nystagmus as well as second and third degree nystagmus.

Massive douching. With the head bent 30° forward, douch the right ear with water at 68° F. for a period of time varying up to 3 minutes. Stop the douching instantly when nystagmus appears. This normally occurs in 40 seconds, and is assumed to be absent if it does not appear within 3 minutes. The nystagmus is rotary to the left. Past-pointing, normally 6 to 8 inches to the right is then tested, following which the head is turned backward 60° from the upright position and the nystagmus again observed, as is also past-pointing with the head in this position. The nystagmus will be observed normally as horizontal to the left. The past-pointing will again be 6 to 8 inches to the right. The patient is then quickly allowed to stand up and a Romberg test is done.

The left ear is then douched in the same manner. The direction of responses will normally be reversed, but nothing else changes. The results are recorded on the Bárány chart.

If the position of the head is 30° forward, the vertical canals of the ear douched are stimulated; with the head 60° back, the horizontal canal is stimulated. In this test these canals are, "minimally" excited according to the laws of nystagmus, but the degree of such minimal stimulation is said to be greater than in the Kobrac test modifications. (The original technique described by Kobrac himself is never used clinically, but always some modification).

SELECTION OF TESTS TO BE USED

1. In a neurological consultation: All rotation tests, massive douching if the drum is intact and the canal is normal.

2. In chronic suppurative otitis media: Fistula test, which alone determines that the labyrinth is viable if the test is positive; 70% cold alcohol douche; rotation tests if the condition of the patient permits.

3. Post-traumatic vertigo: If the drum is perforated, rotation tests only; if the drum is not perforated, rotation tests and massive douching.

4. Ménière's syndrome: Kobrac test; rotation tests if easily tolerated.

NORMALS ABOUT WHICH AGREEMENT MUST BE REACHED

(by a hospital committee appointed for that purpose)

1. Nystagmus after turning 10 times in 20 seconds: 16-24 seconds for horizontal canals; 15-20 seconds for vertical canals.

2. Past-pointing after turning 10 times in 10 seconds: 10-12 inches.

3. Vertigo after turning 10 times in 10 seconds: 16-26 seconds for horizontal canals; 15-22 seconds for vertical canals.

4. Massive douching caloric test with water at 68° F., head 30° forward: Rotary nystagmus after 40 seconds; past-pointing equals 6-8 inches.

5. Kobrac test: Latent period 15-30 seconds in all modifications described above. The duration of the nystagmus is said to vary

from 30-60 seconds to 60-120 seconds depending on modification used.

INTERPRETATION OF TESTS

The interpretation of tests will be done by the house officers only as follows:

1. Normal.

2. "Dead labyrinth" when no response of any kind can be obtained from the labyrinth being tested.

3. Hypofunction of one or more canals when the responses are less than the normals described above.

4. Hyperfunction of one or more canals is to be the diagnosis when the responses are above the normals described.

Any further interpretation of the tests must be done by a member of the visiting staff.

247 COMMONWEALTH AVENUE.

REFERENCES

1. Braun, Alfred: Sinus Thrombophlebitis; Inflammatory Diseases of the Venous Sinuses of the Dura Mater, New York, Paul B. Hoeber, Inc., 1928.

2. Tait, John: The Physiology of the Equilibrial Labyrinth, Medicine of the Ear, New York, Thos. Nelson and Sons, 1939, p. 63.

3. Stevens, S. S., and Davis, H.: Hearing, New York, John Wiley and Sons, 1938.

4. Galambos, R., and Davis, H.: The Response of a Single Auditory-Nerve Fiber to Acoustic Stimulation, J. Neurophysiol. 6:39-57, 1943.

5. Hyndman, O. R.: Physiology of the Vestibular Labyrinth, Arch. Otolaryng. 29:759 (May) 1939.

6. Meltzer, Philip H.: Personal Communication.

7. Fischer, Joseph, and Wolfson, L. E.: The Inner Ear, New York, Grune and Stratton, 1943, p. 127.

BÁRÁNY CHART

(Form used at Massachusetts Eye and Ear Infirmary)

NAME NUMBER

Nystagmus		Spontaneous	POINTING	
Looking to Right		Shoulder from above	Right	Left
Looking to Left				
Looking Up		Nystagmus		
Looking Down		Vertigo		
		Past-Pointing		
		Falling		
		Romberg		
		Turning Head to Right		
		Turning Head to Left		
		Attempt to overthrow		
Head forward 30°		TURNING	POINTING	
To right		Shoulder from above	Right	Left
Amplitude				
Duration	Min. Sec.	Nystagmus		
		Vertigo		
		Past Pointing		
To left		Shoulder from above	Right	Left
Amplitude				
Duration	Min. Sec.	Nystagmus		
		Vertigo		
		Past Pointing		
Head forward 120°		TURNING OR CALORIC	POINTING	
To Right		Shoulder from above	Right	Left
Amplitude				
Duration	Min. Sec.	Nystagmus		
		Vertigo		
		Past Pointing		
		Falling		
To Left		Shoulder from above	Right	Left
Amplitude				
Duration	Min. Sec.	Nystagmus		
		Vertigo		
		Past Pointing		
		Falling		
		CALORIC	POINTING	
Douche Right		Shoulder from above	Right	Left
Amplitude				
After	Min. Sec.	Nystagmus		
		Vertigo		
		Past Pointing		
		Falling		
Head Back			Right	Left
Amplitude				

BÁRÁNY CHART—*Continued*

NAME NUMBER

POINTING

Douche Left
Amplitude — Shoulder from above — Right — Left

After — Min. Sec. Nystagmus
Vertigo
Past Pointing
Falling

Head Back
Amplitude — Right — Left

COCHLEAR
Tuning Forks
Audiometer

X

BRONCHOGRAPHY AS A DIAGNOSTIC AID IN CHEST DISEASE

GEORGE S. MCREYNOLDS, JR., M.D.

AND

FRED W. SHELTON, M.D.

GALVESTON, TEXAS

It has become a generally accepted fact within the past two decades that bronchology is an indispensable part of the proper and intelligent handling of diseases of the chest, more particularly the chronic cases, but also in some instances even the acute ones. However, since there is a large part of the bronchial tree that still is inaccessible to vision through the bronchoscope, we need something more to gain additional information and complete the picture. This can best be accomplished, we believe, by bronchography, using one of the iodized oils as a contrast medium to outline the tertiary bronchi and the peripheral bronchial tree.

In selecting a contrast medium, there are several items that are important. Of course, the first obvious one is that it have the proper iodine content to make it sufficiently radiopaque. The second, important factor is that the oil base have the proper viscosity. If the base is too thin, the medium tends to get out into the alveoli too quickly to permit the taking of roentgenograms. Some workers have the idea that the oil should be warmed before using, which is undoubtedly a good physiological principle, but this tends to lower the viscosity and, again, permits the oil to flow into the peripheral alveoli too rapidly.

Another important factor is the toxicity of the product used. Perhaps the two most commonly used iodized oils employed in outlining the tracheobronchial tree are the products sold under the trade names of Lipiodol and Iodochlorol. Neither of these preparations

---

From the Department of Oto-Rhino-Laryngology of the University of Texas Medical School, Galveston, Texas.

Read before the meeting of the Texas Ophthalmological and Otolaryngological Society, Fort Worth, Texas, December 8-9, 1944.

is known to be toxic and there should be no hesitancy in using them. The only exception might possibly be that in extremely allergic individuals it is possible to bring on an asthmatic attack in rare instances. In the literature, there are references to the development of iodism following the use of iodized oil. Fortunately, we have never seen such a complication. In all of the accompanying studies Iodochlorol was used since it is a cheaper product and apparently as equally efficient as Lipiodol.

The important factor in obtaining good bronchograms is the technique used. Adults and children present two different problems and we will discuss the technique under each group separately.

Technique In Adults. All bronchography in adults is done under local anesthesia. Cocaine solutions can be used, but since pontocaine is so much less toxic we have been using this in a 2% aqueous solution, without epinephrine. These patients should receive, as premedication, atropine and one of the barbiturates. The atropine is important since pontocaine has no inhibitory effect on the secretory action of the mucosal glands as does cocaine, and, in fact, in some instances even seems to stimulate them. This salivation, thus produced, may have a tendency to make the patient cough. The barbiturates are used to offset any toxic effects of pontocaine.

The techinque used in adults is the catheter method, described by Jackson and Bonnier.[1] The pharynx is sprayed with pontocaine. Then the anesthetic solution is instilled, fractionally, a few drops at a time, into the glottis with a laryngeal syringe, under mirror guidance, until the cough reflex is abolished. A soft rubber catheter with a wire stylette is introduced into the glottis after which the stylette is withdrawn and the catheter passed down the trachea. The patient is then taken to the x-ray department where the catheter is manipulated into the proper bronchus under fluoroscopic visualization. The position of the patient is determined by which lobe or lobes are to be outlined. The oil is introduced, again under fluoroscopic visualization. An important point is that only one lung be filled at a time and that both anteroposterior and lateral films be taken to facilitate localization of the lesion.

The following are a few illustrative cases that show not only the results that may be attained with this technique but also demonstrate the value of bronchography in clarifying and completing the diagnosis in pulmonary disease.

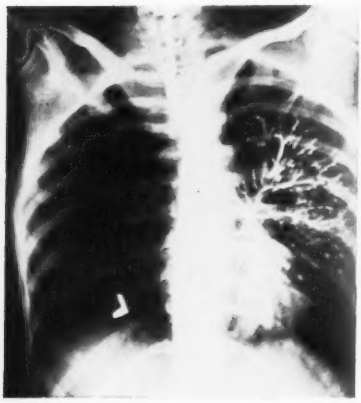


Fig. 1, Case 1.

REPORT OF CASES

CASE 1. C. W. R., a white female, age 23, gave a history of "pneumonia" following an appendectomy about 12 years previous to this examination. Since that time she has had recurrent attacks of "pneumonia" and during the intervals between attacks has had a cough, which was usually productive of varying amounts of foul sputum, accompanied by hemoptysis at times.

The routine chest film showed no particular abnormality. However, at bronchoscopy, purulent secretion was seen coming from the left lower lobe and bronchography demonstrates an extensive fusiform and saccular bronchiectasis of the left lower lobe. This patient receives temporary relief from bronchoscopic aspirations and has not as yet agreed to the suggested surgical extirpation of the affected lobe.

CASE 2. B. D., an 18-year-old white male, also gave a history of "pneumonia" following a surgical operation several years previously, since which time he had continued to have a cough, productive of large amounts of foul sputum, with hemoptysis.

At bronchoscopy, there was such a large amount of foul sputum that it was impossible to be sure that it was all coming from the left lower lobe. However, bronchography showed an extensive, saccular bronchiectasis involving the left lower lobe only. This patient had a left, lower lobe lobectomy and is cured of his disease.

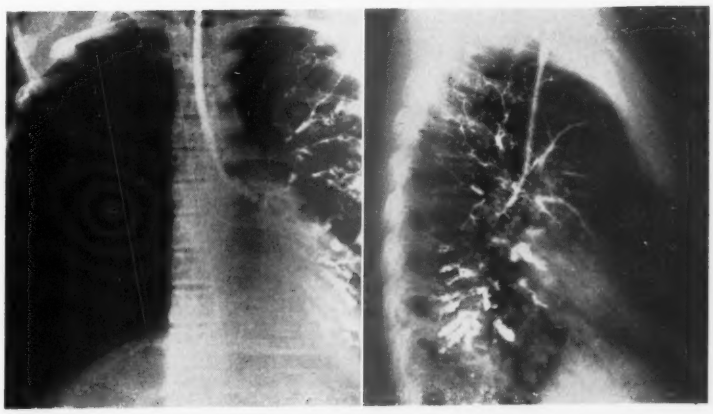


Fig. 2, Case 2.

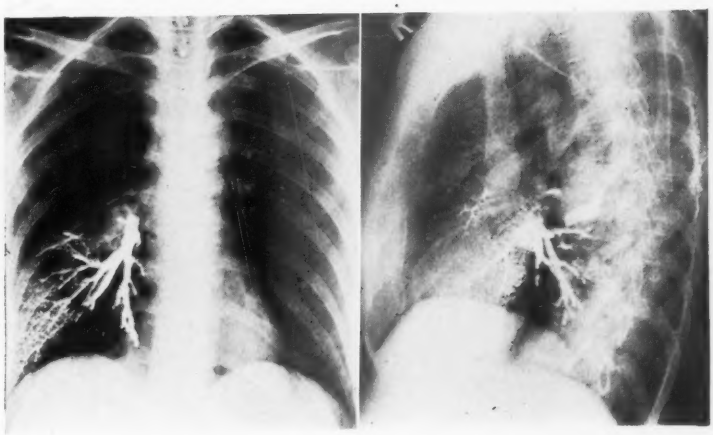


Fig. 3, Case 3.

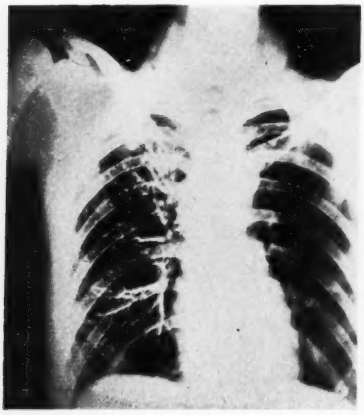


Fig. 4, Case 4.

CASE 3. O. L. C., a 36-year-old white female, was a psychiatric patient. During her hospitalization for psychotherapy, she developed what was thought to be influenza. Following this, she continued to have a productive cough and, on several occasions, hemoptysis.

Routine roentgenray examination of her chest showed only an area of increased density near the right hilum which was not diagnostic in appearance. However, bronchography showed a fairly large abscess cavity in the apical portion of the right lower lobe. She was advised to have surgery after she had failed to improve under bronchoscopic treatment, but, unfortunately, she passed out of our care and no follow-up is available.

CASE 4. M. M., a 42-year-old white female, had been treated for active tuberculosis by pneumothorax. Her general condition, as indicated by gain in weight, afebrile state, negative sputum, and normal sedimentation rate, indicated an arrest of her disease. However, she continued to have a productive cough and was, consequently, still considered as potentially tuberculous.

With bronchography, we were able to demonstrate a cylindrical bronchiectasis of the right upper lobe, presumably secondary to the fibrotic healing of the tuberculous infection.

CASE 5. B. M., a 30-year-old white female, gave a history of prolonged cough, hemoptysis, and fever before she was bronchoscoped,

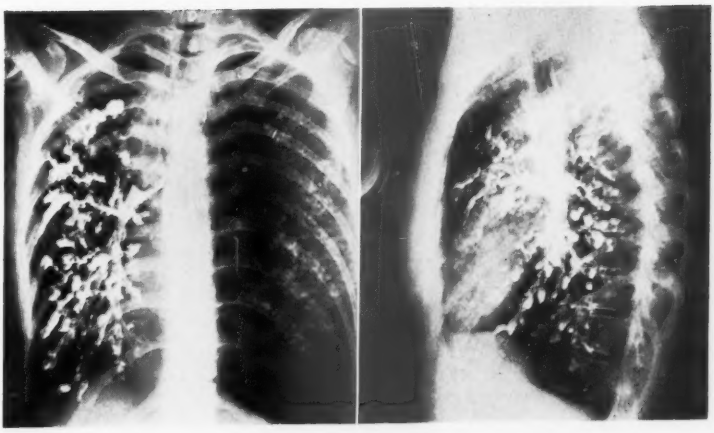


Fig. 5, Case 5.

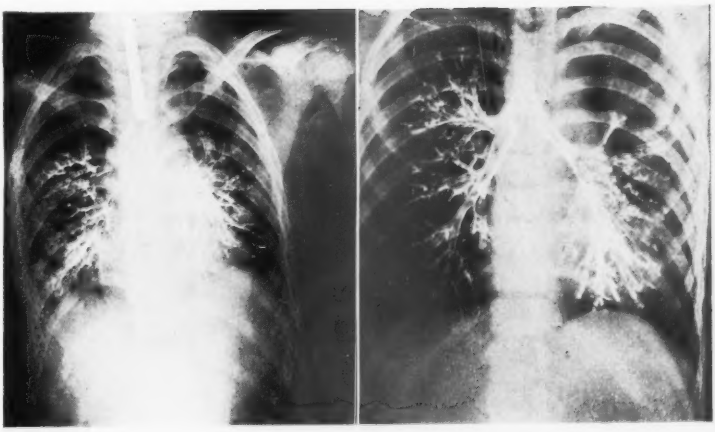


Fig. 6, Case 6.

Fig. 7, Case 7.

finally, in 1939. At that time a tumor was visualized, obstructing the right main bronchus. This obstructing tumor was removed endoscopically after being diagnosed "adenocarcinoma," following which she received irradiation therapy.

She was first seen by us in 1942, at which time she complained of a productive cough, with blood streaking at times. These episodes were repeated at varying intervals. Bronchography demonstrated the extensive bronchiectasis of the entire right lung as well as outlining the tumor protruding into the right main bronchus. Because of the extensive bronchiectasis, and not because of the tumor, which has been proven to be benign adenoma, she was subjected to a total pneumonectomy from which she made an uneventful recovery.

Technique In Children. There have been many techniques dedescribed for use in children, since it is not possible to obtain satisfactory local anesthesia in uncooperative patients. In consultation with our Departments of Pediatrics and Anesthesiology, we decided to try iodized oil instillations under general (ether) anesthesia. The patients are prepared in the usual way for a general anesthetic. They receive morphine and atropine or scopolamine as a premedication. After gas-ether induction, open drop ether is administered, on the fluoroscopic table, until they are in deep third stage anesthesia. At this stage, the intercostal muscles are eliminated from the respiratory effort and breathing is carried on by the diaphragm alone. Then the anesthetic is stopped. The larynx is exposed with a laryngoscope and the bronchoscope is introduced. A flexible-tipped bronchial aspirator is passed through the bronchoscope and any secretion in the bronchial tree is aspirated. Then the syringe containing the iodized oil is connected to the aspirator and the oil is injected, under fluoroscopic visualization, into the desired lobe or lobes. The child is already on the x-ray table so no moving is necessary before taking the films.

In children, we usually fill both the right and left bronchial trees at one time to obviate the necessity of repeating the general anesthetic. However, one must be guided in this by the amount of pulmonary pathology present in the individual case. There are some cases with extensive pulmonary suppuration in which it would be inadvisable to fill both sides at one time. When both lung fields are outlined, lateral films are of no value, but oblique films are of great advantage.

With the patient in third stage anesthesia, the cough reflex is abolished and the breathing is slow and regular so that it is possible to get excellent films. It should be pointed out that satisfactory films

Fig. 8, Case 8.

can be obtained only when the patient is either completely awake and cooperative or is in deep anesthesia. Also, one must have the complete cooperation of a competent anesthetist, not only for the procedure to be safe, but also to secure satisfactory roentgenographic studies.

Contraindications. Of course, objections will be raised as to the danger of giving an inhalation anesthetic to a patient with suspected, or known, suppuration. This seems even more important to us than it may to some of you since all our endoscopy in children is done without any anesthesia except, in some cases, preoperative sedation. However, we have used this technique in some 20 children in the last year and one-half without any discernible ill effects whatsoever. Several of these patients have had such extensive suppuration that total pneumonectomy was required to effect a cure. We feel that the aspiration of the bronchial tree before instillation of the iodized oil is an important factor in avoiding complications in using this method.

There are relatively few contraindications to the use of this technique in children. Of course, it should not be used in acutely ill children. However, practically all these patients are chronic chest cases who are being prepared for thoracic surgery. Also, it has been

rather generally accepted that iodized oil should not be used in the acute exudative forms of pulmonary tuberculosis. Nevertheless, the present trends in endoscopy in pulmonary tuberculosis tend to dispel the fears of using iodized oil in suspected or even known cases of acid-fast infections.

REPORT OF CASES

CASE 6. W. H. S., a 10-year-old white male, had had an empyema of the right pleura drained about two years before and had, apparently, recovered. Then several months before admission he had a recurrence of swelling at the site of the previous drainage. This was opened and continued to drain. Iodized oil was instilled in the bronchial tree to rule out the possibility of bronchiectasis or lung abscess. This showed only a mild, cylindrical bronchiectasis having no connection with the draining sinus, which was later proven to be an osteomyelitis of the rib.

CASE 7. G. T., a 10-year-old white male, was admitted with a complaint of recurrent attacks of cough and pain in his left chest, following pneumonia four years previously. The routine chest examination, both clinically and roentgenographically, was essentially negative. However, bronchography showed a comparatively mild bronchiectasis, of the cylindrical type, in the left lower lobe, which was apparently the site of his previous pneumonia. This was not considered to be serious enough, at the time, to warrant surgery.

CASE 8. A two-year-old colored male had been considered, since birth, as having a congenital atelectasis of the right upper lobe. However, after the instillation of iodized oil, I believe that we have shown that this shadow adjacent to the upper mediastinum is not the result of an upper lobe atelectasis.

CASE 9. D. L., a two-year-old white male, was admitted with a history of possible foreign body and showing an atelectasis of the right lower lobe. No exogenous foreign body was found and, after several bronchoscopic aspirations, the atelectasis cleared to some degree and the patient was asymptomatic. However, bronchography shows that he still has a carnified lower lobe, with bronchiectasis.

CASE 10. S. P., a four-year-old white female, had had a cough, fever, and hemoptysis for the past two years, following an attack of pneumonia. The instillation of iodized oil showed that she had a rather extensive bronchiectasis limited to the right lower lobe. A lobectomy was done and the child made a nice recovery.

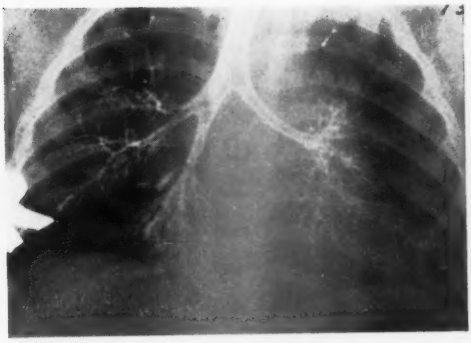


Fig. 9, Case 9.

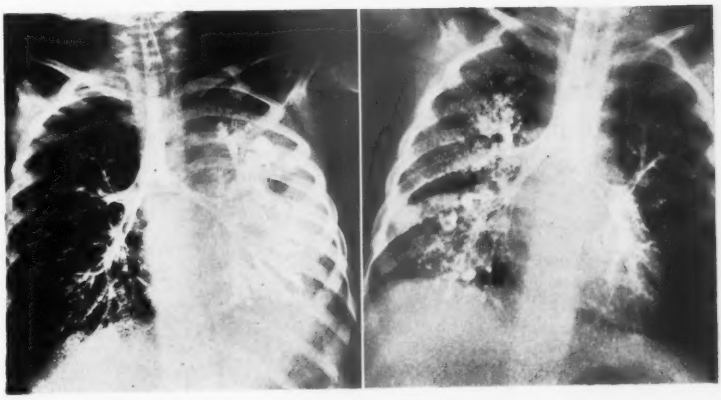


Fig. 10, Case 11.

Fig. 11, Case 10.

CASE 11. J. W., a six-year-old white male, was admitted with a history of fever, cough, and hemoptysis, which began ten months previously.

From the routine chest examination, it is obvious that he had an extensive suppurative process of the left lung, but after the instillation of iodized oil we had a much more graphic and complete demonstration of the tremendous amount of bronchiectasis present. A pneumonectomy was done and the child made an excellent recovery.

COMMENT

We have described two methods of bronchography, the one for use in adults, with local anesthesia; the other for use in children, with general anesthesia. Also, we have selected a few cases of each group which represent the different chest conditions we more commonly see.

It is just as important to use bronchography to rule out various bronchopulmonary lesions as it is to definitely establish the presence of pathology. Also, it is particularly important to outline the supposedly normal portions of the lungs in a patient with a pathological condition before deciding on the type of treatment to be undertaken.

Bronchography should not be looked upon as a substitute for bronchoscopy in any case, but as an adjunct to bronchoscopy. We believe, and insist, that in almost every case a diagnostic bronchoscopy should precede the instillation of the iodized oil.

UNIVERSITY OF TEXAS MEDICAL SCHOOL.

REFERENCES

1. Jackson, C. L., and Bonnier, M.: Technique of Bronchography, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 46:771 (Sept.) 1937.

2. Jackson, C. L., and McReynolds, G. S.: Anesthesia for Peroral Endoscopy, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 49:1048 (Dec.) 1940.

3. Field, C. E.: Bronchography in Children, Lancet 2:357-359 (Sept. 26) 1942.

4. Baker, A. H. L.: Endobronchial Lipiodol Under General Anesthesia in Children, Brit. J. Anesth. 17:112-116 (Jan.) 1941.

XI

INDIVIDUALIZATION IN THE MANAGEMENT OF CARCINOMA OF THE MAXILLARY SINUS

MAURICE F. SNITMAN, M.D.

CHICAGO, ILL.

The site and direction of extension of carcinoma of the antrum have definite prognostic significance. The prognosis has been influenced also by an increasing knowledge of radiation therapy especially as it pertains to its technical application. One of the main objectives of this paper is to call attention to a plan of management suggested by Dr. Danely P. Slaughter, Director of the Tumor Clinic at the University of Illinois College of Medicine. This plan consists of the application of radium within the cavity as a supplementary measure to external protracted irradiation, after exposure of the antrum by conservative surgical intervention. The results of this technic have been highly satisfactory and have obviated the necessity of radical operation.

The rationale of treatment of most neoplasms is based upon the fact that they originate and exist as a localized process for a prolonged period before dispersion takes place. During this early localized stage, cure is possible by total destruction of the growth. The ideal situation is one in which an early diagnosis has been made. With reference to carcinoma of the maxillary sinus it is depressing to note how often the patient has suffered pains in the teeth or paresthesia with or without infra-orbital pain for prolonged periods before seeking medical advice. Nearly as often do we find patients, after having at last consulted a physician, spending additional time for the extraction of teeth from the upper jaw without benefit. No small number of patients are also subjected to antral lavage or minor intranasal surgical procedures for the relief of "chronic sinusitis." It is chiefly because of these facts that it is rare to find a malignant tumor of the antrum before it has appeared beyond the confines of the antrum cavity. A very early cancer that could be removed surgically is

---

From the Department of Laryngology, Rhinology and Otology and the Tumor Clinic, University of Illinois College of Medicine.

Read before a meeting of the Illinois State Medical Society, Section on Ear, Nose and Throat, Chicago, Ill., May 17, 1944.

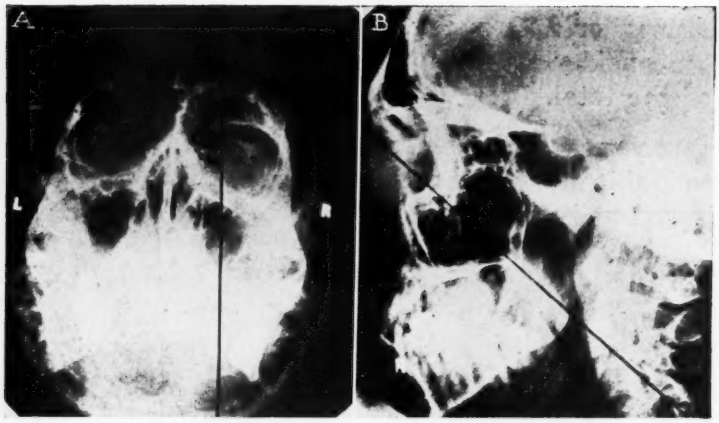


Fig. 1.—Malignancy planes of the maxillary sinus as suggested by Ohngren. A. A vertical plane divides the antrum into medial and lateral halves. B. The antrum is further subdivided into anterior and posterior portions by an oblique plane passing through the inner canthus and angle of the mandible.

usually accidentally recognized during operative treatment for benign sinusitis. A more comprehensive study of patients presenting pains in the trigeminal area and unilateral rhinitis after the fourth decade may lead to the more timely application of treatment.

The early treatment of carcinoma of the antrum was entirely surgical. The results were uniformly bad because the growths were well advanced when first seen and it was almost impossible to circumvent the lesion; as a consequence recurrence was the rule. Since the effects of radiation on neoplastic disease have become known, better results have been reported from the combined use of radiation and surgery. The irradiation was obtained from the placement of radium in the antrum following surgical exposure. An added factor of the increased cure-rate was the employment of electrosurgery. Statistical reports of large series, such as those of New[1, 2] and Ohngren[3] show five-year cures in 30 to 50 per cent of all types of malignancies of the maxillary sinus.

Through the pioneer experimental work of Regaud and its clinical application by Coutard,[4] a great advance in roentgentherapy was revealed by the divided dose principle. The cure-rate of carcinoma of

many regions increased perceptibly. Since this method became popular in 1931, two communications have appeared in the literature dealing with carcinoma of the antrum. Watson[5] reported a series of 73 cases with five-year cures in 15 (20.5 per cent). Every cured patient during the last seven years had received external irradiation. In six instances this form of therapy was the only treatment employed. Del Regato[6] treated 10 inoperable cases by the sole use of external irradiation and obtained 5- to 15-year cures in 4. It may be reasonable to assume that more favorable reports of this form of therapy should follow, if we are to judge by its effectiveness on cancer elsewhere.

In spite of the advanced condition of the cancer of the maxillary sinus when first seen, the location of the bone destruction permits a general division into two chief malignancy areas, as suggested by Ohngren.[3] A line, extending from the inner angle of the eye to the angle of the jaw, divides the antrum into a suprastructure and an infrastructure (Fig. 1). The comparative cure rate of these two areas is great and a regional classification should be attempted in case reports, if one is to obtain significant information from a therapeutic standpoint. Unfortunately, the results submitted in most publications are scarcely a sound measure of the capacity of a given treatment to cure maxillary sinus tumors, since little attention is paid to the site of origin.

Lesions above the Ohngren malignancy plane, situated medially, are frequently associated with nasal polyps, and encroach on the meninges more readily and tend to early lymphatic invasion. In the lateral part of the suprastructure, carcinoma has at its onset the most dormant development. It then rapidly invades the malar bone producing the characteristic tumefaction of the external angle of the floor of the orbit.

Malignancies in the infrastructure of the antrum produce earlier symptoms referred to the teeth, but these symptoms are still overlooked until late because attention is directed to the dental structure for a considerable period of time.

In the consideration of a therapeutic regime, it is essential to consider two chief factors, the site of the primary lesion and the presence or absence of lymphatic extension. The element of radiosensitivity of antral carcinoma can only be gauged by the responses of the cancer in general, since protracted fractionated irradiation has as yet not received sufficient trial. Lesions involving the roof of the sinus present a most unfavorable prognosis, and it is unfortunate that this area

Fig. 2. Case 1.—External evidence of malar bone destruction was shown by a firm swelling below and lateral to the left eye. No palpable cervical nodes.

is the affected site in most cases. Surgical treatment would require mutilating procedures with sacrifice of the eye in many cases, if there is a promise of a complete cure. In spite of the recognized therapeutic advances arising from the combination of electrosurgery and local radium implantation, there is much to be desired. This, notwithstanding the good results reported by New[1, 2] since he states that the patients selected for treatment were those who appeared to have a reasonable chance of being cured. The determination of the extent of regional invasion beyond the confines of the antral walls is not feasible, to permit circumventing the cancer by surgical measures. In the absence of definite proof, we may not state whether, in the cured cases, electrosurgical removal or the effects of radium implantation was the deciding factor. It would be logical to favor the latter, in view of the more recent reports of cures by employment of

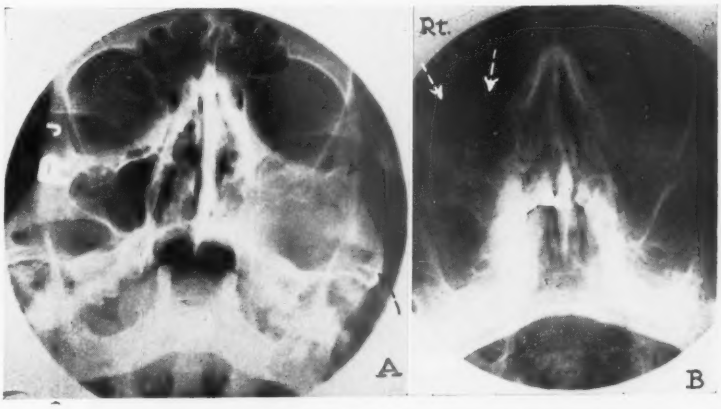


Fig. 3.—A. Roentgenogram of Case 1, illustrating malar bone destruction without disfiguration of orbital floor. The left antrum is cloudy. B. Roentgenogram of Case 2, showing cloudiness of right antrum and destruction and bulging upwards of the orbital floor.

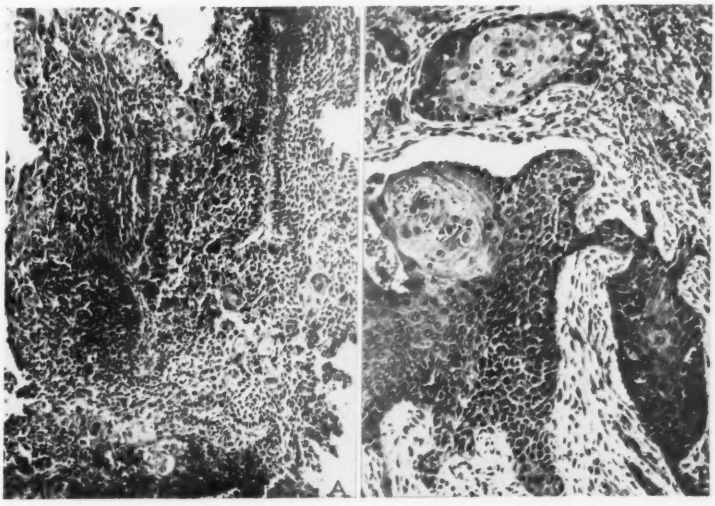


Fig. 4.—A. Biopsy, Case 1—cylindric cell carcinoma with squamous cell metaplasia. B. Biopsy, Case 2—well differentiated squamous cell carcinoma with keratinization and tendency to pearl formation.

Fig. 5. Case 1.—Large sublabial fistula which permits direct observation of the antrum. This fistula is kept closed by a dental plate prothesis.

protracted external irradiation.[5,6] Mutilating radical surgery is becoming less prevalent as our knowledge of radiotherapy increases. What benefit may accrue from the added supplementation of radium placed in the sinus, as practiced in the cases reported in this paper, will naturally have to await further observation.

Lesions in the floor of the sinus with involvement of the hard palate and alveolar ridge are amenable to intra-oral surgical removal, followed by local radium application. At present, no statistics are available to show in what percentage of cases recurrences can be avoided by the supplemental use of external protracted irradiation.

Treatment of the primary lesion is not affected by the presence of regional metastases. If metastases are present, a new therapeutic problem presents itself. Carcinoma of the head and neck, if untreated, produces metastases to the cervical lymph nodes. The latter serve as protective barriers to the spread of cancer, and Crile, years ago, called attention to the extensive collar of lymphatics about the

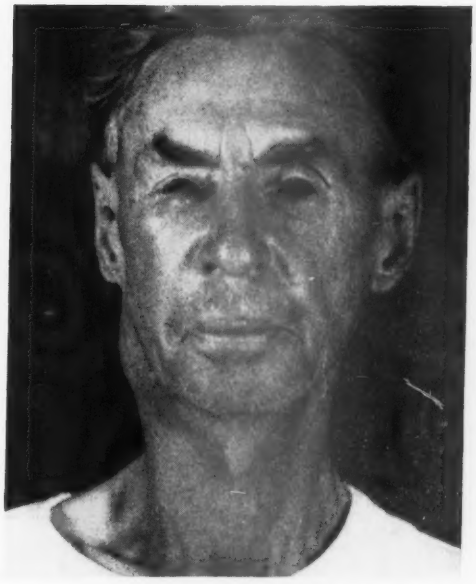


Fig. 6. Case 2.—The narrowing of the right palpebral fissure, swelling of the malar region and large upper cervical node are well illustrated.

neck and its effectiveness in preventing spread below the clavicle. In this connection, it is interesting to note that Braund and Martin[7] found distant metastases in only 23 per cent of a series of 284 autopsies, where death resulted from cancer of the upper respiratory and alimentary tract. Death usually results from the effect of the primary and cervical node involvements, producing sepsis, hemorrhage, inanition or pneumonia from aspiration.

Patients with cancer of the head and neck present one of three conditions in the cervical region: 1. no palpable metastastic nodes; 2. operable nodes; 3. inoperable nodes.

The management of the nodes of these groups at the Tumor Clinic of the University of Illinois follows closely the plan as outlined at the Memorial Hospital, New York City. In brief, no prophylactic dissection or irradiation is performed. If the nodes are operable and the control of the primary lesion is assured, neck dissection is performed, providing the requirements laid down by

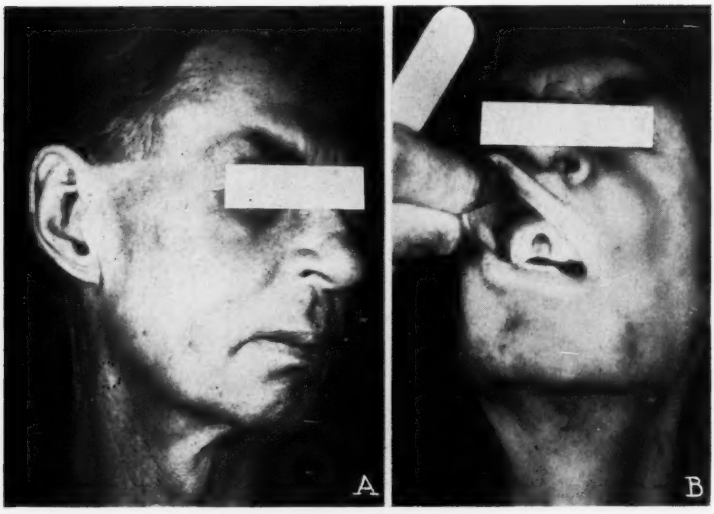


Fig. 7. Case 2.—Photograph of patient taken in March 1944. A. The cervical node enlargement is replaced by a fibrotic nodule. The swelling of the malar region has disappeared. B. Sublabial fistula permitting direct observation of the antrum.

Duffy[8] are met. If the nodes are inoperable, the patient is submitted to external radiation supplemented with radon seed implantation.

REPORT OF CASES

I shall cite two cases not because cures are at present claimed for them but merely to illustrate more specifically the program of treatment which seems to hold out promise of improved results over the former plans of management.

CASE 1. A. R., white female, aged 42 years. When first seen in April 1943, this patient complained of bloody expectoration for a period of four weeks. In 1937, there was a numbness of the left side of the face, postnasal discharge and frequent sore throats. There followed severe headaches and frequent recurrences of upper respiratory tract infection. In June 1942 all teeth were extracted. In February 1943 the patient suffered from acute exacerbation of upper respiratory tract infection with consequent nasal obstruction, bloody expectoration and redness of the left eye. She complained of a sensation of crawling over the left cheek.

On examination, the nose was essentially negative. There was a distinct swelling over the left cheek, below and lateral to the eyes, which seemed to limit its lateral movement (Fig. 2). The left antrum was dark on transillumination and yielded a negative return on irrigation. Roentgenologically, over the left malar area, there was increased density with evidence of definite change in the bone itself (Fig. 3). There was a suggestion of a tumor mass, but no cervical metastases could be discerned. On April 28, 1943, the antrum was opened sublabially and a mass filling the cavity was observed. Biopsy of this tissue revealed a cylinder cell carcinoma with a fair degree of squamous cell metaplasia (Fig. 4A).

Since the patient refused further surgical intervention, external irradiation was begun on May 24, and until June 26, 4800 r were given to two portals over the involved area. The radiation factors were 3x3 cm. portals, 200 kilovolts, filter ½ cu. plus 3 al, target-skin distance 50 cm., using a daily dose of 400 r. On July 1, after further surgical intervention was consented to, the external carotid artery was ligated and the antrum reopened. The greater part of the canine wall and the alveolar process were removed. On July 30, 1500 hrs. of radium were applied to the antral cavity. The healing was uneventful with a permanent antral fistula. The method of radiation consisted of inserting radon seeds in the lumen of the antrum and leaving them in situ for 64 hours. Examination in April 1944 disclosed a smooth-lined cavity with no evidence of disease or cervical adenopathy (Fig. 5). There has been no change at the time of this writing.

Case 2. F. W. H., male, age 67. This patient was first seen in October 1943, with complaint of a swelling of the right cheek and narrowing of the palpebral fissure (Fig. 6). He also had a large swelling of the right upper cervical node (substernomastoid) and a smaller one in the lower part of the posterior triangle. The duration was 14 months when medical examination was first sought. Health was good until this time, but during the past few months there was a loss of weight of ten pounds. Clinical examination was negative, but x-ray films revealed an involvement of the malar bone and the floor of the orbit. The antrum was cloudy. There was node involvement as previously described.

On October 15, the right external carotid artery was ligated and the antrum on the same side was exposed sublabially. The cavity was filled with friable degenerated tissue, which on histologic study showed a well-differentiated squamous cell carcinoma (Fig. 4B). Erosion was limited to the malar bone and the floor of the orbit. The antero-

lateral surface of the sinus was removed and the cavity cleared of degenerated tissue. Seven radium needles, each 10 mg., were arranged in a circle in the cavity and protected from the bone by 1 cm. of vaseline gauze. The radium was left in situ 28½ hours, yielding a dosage of 2000 mg. hrs. of radiation. Into the upper cervical node 13 radon seeds (2600 MCH) were implanted directly through the skin. Metastatic involvement of the cervical nodes was confirmed by aspiration biopsy. The lower node received 4 radon seeds (800 MCH). On November 10, 1943, 7 more radon seeds (1350 MCH) were implanted into the upper node, and 10 days later an epidermite developed in the area of this node. From November 3 to December 16 the patient received external roentgen radiation to the antral area through two portals until a total dose of 5260 r. was given. The radiation factors were: 5x6 cm. portal, 140 kilovolts, filter ¼ cu plus 1 al., target-skin distance 40 cm., using a daily dose of 150 r. From December 18, 1943, to January 12, 1944, he received another series of treatments to the right side of the neck for a total dose of 3800 r, using 12x15 cm. skin portal with a daily dose of 200 r.

In the latter part of March 1944, examination disclosed a fibrotic nodule replacing the upper cervical node (Fig. 7A). The antrum fistula permitted free inspection of the cavity which showed a fair degree of radionecrosis, but no residual lesion was observed (Fig. 7B). In April 1944, the patient refused further medical attention and subsisted exclusively on alcohol, expiring from inanition.

SUMMARY AND CONCLUSIONS

1. The site of origin of carcinoma of the maxillary sinus can in many instances be ascertained. Due consideration of this factor is of great value in the determination of the program of management and its therapeutic evaluation.

2. Attention is called to the value of radium placed in the sinus as a supplement to external protracted irradiation after conservative surgical intervention.

3. Protracted fractionated irradiation appears to be an extremely favorable adjunct in the treatment of antrum carcinoma.

4. While our therapeutic weapons have not changed in recent years, our end results have improved because we are learning to apply them with greater effectiveness.

1853 West Polk Street.

REFERENCES

1. New, G. B.: Malignant Tumors of the Antrum of Highmore; End-Results of Treatment, Arch. Otolaryng. 4:201-214, 1926.

2. New, G. B., and Cabot, C. M.: The Curability of Malignant Tumors of Upper Jaw and Antrum, Tr. Am. Laryng., Rhin. & Otol. Soc. 41:584-590, 1935.

3. Ohngren, L. G.: Malignant Tumors of the Maxilloethmoidal Region: Clinical Study with Special Reference to Treatment with Electrosurgery and Irradiation, Otolaryn., Supp., 19:1, 1933.

4. Coutard, H.: Results and Methods of Treatment of Cancer by Radiation, Ann. Surg. 106:584-598, 1937.

5. Watson, W. L.: Cancer of Paranasal Sinuses, Laryngoscope 52:19-23, 1942.

6. Del Regato, J. A.: Roentgentherapy in Epitheliomas of the Antrum, Surg., Gynec. & Obst. 65:657-665 (Nov.) 1937.

7. Braund, R. R., and Martin, H. E.: Distant Metastasis in Cancer of the Upper Respiratory and Alimentary Tracts, Surg., Gynec. & Obst. 73:63-71 (July) 1941.

8. Duffy, J. J.: Cervical Lymph Nodes in Intraoral Carcinoma: Surgery or Irradiation? Am. J. Roentgenol. & Radium Ther. 39:767-777 (May) 1938.

XII

THE SIGNIFICANCE OF HOARSENESS

WAITMAN F. ZINN, M.D.

BALTIMORE, MD.

My purpose in bringing this subject before you is to emphasize the prime importance of hoarseness, alone or accompanied by other symptoms, as a warning signal of "danger ahead." I feel very strongly that the significance of hoarseness has not been given the recognition it deserves in view of the frequency with which this symptom is encountered in various diseases and the seriousness of the conditions which follow in its wake. This is undoubtedly due, in part, to the fact that nearly everybody, at one time or another, has been hoarse from minor ailments and has recovered uneventfully. It is the duty of the medical profession to educate the public concerning hoarseness and arouse them to the dangerous sequelae of this symptom.

First of all, what is hoarseness? It has been very aptly defined by Frank[1] as "any alteration in the speaking voice which results in a roughened or rasping character to the voice." This alteration may be produced by impairment of the function of any one of the several parts that go to make up the speech mechanism. This latter is composed of the larynx (or voice box), in which are located the vocal cords, which are set in vibration by a column of air from the lungs, this molded into speech by the tongue, teeth, lips and palate. The vocal cords are tensed by the laryngeal muscles (those particularly concerned being the thyro-arytenoideus, the crico-arytenoideus lateralis and the interarytenoideus), and the lumen of the glottis is narrowed to provide resistance to the ascending air current. The edges of the stretched cords are forced open, the air escapes, lowering the pressure in the trachea and allowing the cords to again approximate, resulting in an instantaneous rise in pressure and a repetition of the entire process. It is this rhythmical production of puffs of air that results in sound. It must be borne in mind that the lungs, and the cavities of the mouth, nose, and pharynx all contribute to speech production. In addition to this, the larynx, the lungs and the pharynx are further dependent on nervous control.

Presented before the Southern Section of the American Laryngological, Rhinological and Otological Society, Charlotte, N. C., Jan. 15, 1945.

This brief review of speech mechanism will serve to emphasize the possibilities presented by a case of hoarseness. It can be seen that such a symptom may point the way to a disease far removed from the larynx though affecting the latter secondarily.

What, in general, are the causes for hoarseness? The laryngologist is familiar with a large number of diseases both local and systematic which tend to alter the tone of the normal speaking voice. These conditions should be presented to the general practitioner who is usually the friend and advisor of the families he attends. The internist must be cognizant of the dangers faced by his patient and he should insist that the advice of the consulted laryngologist be carried out. Hoarseness may result from the following conditions:

1. Inflammatory infections of the larynx—the more common being acute and chronic laryngitis, tuberculosis, lues, diphtheria, influenza, measles and scarlet fever;

2. Trauma—including all types of wounds to the larynx, internal and external;

3. Tumors—benign and malignant;

4. Disturbances of the central and peripheral nervous systems—including aneurysms, brain tumors, and any condition producing pressure or injury to the recurrent laryngeal or vagus nerves, and hysterical aphonia.

As a word of warning, let me say that functional hoarseness should not be diagnosed until all organic lesions have been excluded. This condition requires as careful study and treatment as if a definite lesion were present.

Of the cases of hoarseness examined by me, tumors, or neoplasms, either benign or malignant, have been found in the largest number of cases. As pointed out by Jackson, the consideration of extrinsic and intrinsic growths is not as applicable in diagnosis as in treatment. The hoarseness depends upon the involvement of the motor mechanism (the cord, the arytenoid cartilage, the crico-arytenoid joint, and the nerves and muscles concerned in their motility).[2] It depends, in other words, on the location of the tumor, and whether or not it interferes with normal laryngeal movements. The removal of benign tumors should restore the voice to normal unless there has been damage to the underlying tissues at the time of operation, or previously. Jackson and Jackson have noted a tendency to phonate with the ventricular bands following recurrence of papilloma, though this is uncommon, not

having been generally recognized by laryngologists and accounting for an estimated 4 per cent in the cases of hoarseness encountered by them. They go on to state that "in a well-established case of phonation with ventricular bands the voice is rather deep in pitch and more or less rough in quality. In the early stages, the patient complains that his voice 'breaks' and he can never be sure of the pitch. Sometimes two tones are produced at once. If this condition is recognized early, full development can be prevented by a logopedic instructor".[2] Again, they state that "a loud rough voice was produced by vicarious action of the ventricular bands and not by the vocal cords which were propped apart by a tumor mass".[2]

I recall the case of a young man, aged 39, who was sent to me with the only symptom of hoarseness, extending over a two and one-half year period. His tonsils had been removed and the antra punctured and irrigated numerous times with no improvement in his voice. Laryngeal examination revealed a pedunculated growth attached to the anterior third of the right cord. Removal completely restored his voice and the pathological report was, fortunately, benign papilloma. The patient reported that he had not had a laryngeal examination previous to being referred to me.

It is now pretty generally conceded that 80% of all cases of carcinoma of the larynx are curable by operation if the diagnosis is made while the disease is still intrinsic. In a series of 25 cases of total laryngectomy reported by me in 1938, 20 (or 80%) gave a history of preoperative hoarseness for periods varying from six months to over two years, and of these 20, 16 (or 80%) were cured by operation (Table 1). In another series of 178 cases, hoarseness was a constant symptom, but 100 of these patients were not referred until all classical symptoms of carcinoma were present and they had to be classed as inoperable. Of 144 cases of carcinoma of the larynx recently examined by me, 134 (or 93%) gave a history of hoarseness as a first symptom. Jackson and Jackson report statistics on 410 cases in which hoarseness was an early symptom in 95% of the cases.

Smith,[3] in connection with a series of cases of laryngectomy reported by Lewis, found that in 100 patients an average of 11½ months had elapsed between the onset of marked hoarseness and the diagnosis of carcinoma of the larynx. The delay affected unfavorably the prognosis of laryngectomy. The practice of prescribing some remedy for hoarseness before a definite diagnosis is made cannot be too strongly discouraged.

TABLE 1.

25 CASES REPORTED IN "LARYNGECTOMY—ONE STAGE"[12] SHOWING LENGTH OF TIME OF HOARSENESS AND THE RESULTANT CURE AFTER TREATMENT

TIME						RESULTS
Unknown	6 mos.	9 mos.	12 mos.	18 mos.	2 years	over 2 years	
4						3 cured 1 died 75%
	6					5 cured 1 died 83.3%
		5				4 cured 1 died (other cause) 80%
			3			2 cured 1 died 66.6%
				1		cured—100% died—heart
					1	cured—100%
					1	cured—100% 8 yrs.

Of a total of 25 patients, 20 (80%) gave a history of hoarseness; of these 20, 16 (80%) were cured.

It must be pointed out that it is quite possible to have a carcinoma of the larynx without any hoarseness present. This would, of course, be due to the location of the lesion and the fact that it did not interfere with the phonatory action of the cords. However, I agree with Jackson when he points out that it would be possible to pick up even this small percentage of cases in the intrinsic stage were a practice made of including a thorough laryngeal examination for any complaints referable to ear, nose or throat.

In 1938, Negus[4] presented a paper on the subject of hoarseness emphasizing its importance as "an early symptom of some progressive

disease which may kill the patient if not arrested." His discussion of the causes was presented in the form of age levels: in children—diphtheria, acute and chronic laryngitis, vegetable foreign body, papillomata; in young adults—repetition of any conditions found in childhood with the addition of simple chronic laryngitis (from voice strain), singer's nodes, fixation of the crico-arytenoid joints following early diphtheria or a high tracheotomy, and laryngeal tuberculosis (usually secondary to a pulmonary condition). The prognosis in a case of pulmonary tuberculosis is worse if the larynx is affected. In adults the causes of hoarseness are listed as inflammatory diseases (chronic and acute) including irritation of the cords from tobacco, alcohol or overuse; syphilitic laryngitis, malignant neoplasms, simple neoplasms, and neurological disorders. Hoarseness in women is most often caused by functional aphonia and postcricoid carcinoma. In elderly men, the condition most likely to be found is intrinsic carcinoma of the larynx. Negus urges that no patient should be continuously hoarse for more than three weeks without laryngeal examination and diagnosis.

In 1937 a suggestion was made by LeJeune[5] which deserves more attention than it has apparently received up to the present time. The suggestion was that laryngologists set up a program of education of the general practitioner and of the public, making them "voice conscious" and "hoarseness conscious." It was pointed out that the general surgeon has done a similar job over the years in educating women to the dangers and significance of a lump in the breast, and as a result has decreased the mortality from cancer of the breast considerably. Hoarseness should be regarded as nature's first signal of distress and should indicate an immediate and thorough diagnosis and treatment.

Following in LeJeune's footsteps, Mitchell[6] urges a program of public education, pointing out the advantages of early diagnosis and the dire results of neglect.

In our eagerness to make the earliest possible diagnosis of carcinoma of the larynx, we must not overlook the fact that the three diseases most responsible for hoarseness, cancer, tuberculosis and lues, can all be present at the same time in the larynx, and the discovery of one does not rule out either of the other two. The diagnosis must be exhaustive and complete.

I have not up to this point said much about tuberculosis or lues as a cause of hoarseness. Laryngeal tuberculosis is usually secondary to the pulmonary condition and is a late development of the disease. Not many, but a few cases of pulmonary tuberculosis are first diag-

nosed because of an investigation of hoarseness (the only symptom). I recently examined a patient in our bronchoscopic clinic whose history and apparent symptoms indicated carcinoma but whose final diagnosis was advanced tuberculosis. Hulse[7] reports that "hoarseness is the most consistent symptom in tuberculosis of the larynx, yet the percentage of patients with hoarseness who have specific laryngeal involvement is small." In syphilis, as in tuberculosis, the lesion is rarely primary and diagnosis is made by supportive tests.

In a most thorough discussion of the subject of the significance of hoarseness, Wells[8] added an interesting note in reviewing the fatal illness of our first president, whose one constant and outstanding symptom in his brief and tragic illness was hoarseness. He concluded that death was no doubt "due to an intense streptococcic inflammation of the larynx attended with edematous swelling, which closed the narrow opening behind the vocal cord causing suffocation." Such a consideration should add "to our thoughtful respect of hoarseness as a symptom."

In conclusion, let me urge the persistent and untiring support of a program of education for the general practitioner and the public on the prime importance of hoarseness as a warning of coming danger. In lectures to medical students, stress the necessity of careful, not casual examination of the larynx. Insist that all students learn to make full and complete examinations and diagnoses guided by their findings. Let's reduce death from laryngeal disease by early diagnosis and treatment.

MEDICAL ARTS BUILDING.

REFERENCES

1. Frank, D. I.: Hoarseness—A New Classification and a Brief Report of Four Interesting Cases, Laryngoscope 50:472-478 (May) 1940.

2. Jackson, Chevalier, and Jackson, C. L.: Cancer of the Larynx, Philadelphia, W. B. Saunders Co., 1939.

3. Smith, A. J.: Cancer of the Larynx, Pennsylvania M. J., p. 258, Aug., 1932.

4. Negus, V. E.: The Signficance of Hoarseness, N. Y. State J. Med. 39:9-13, 1939.

5. Le Jeune, F. E.: Hoarseness, Surg., Gyn. and Obst. 66:405-408 (Feb.) 1938.

6. Mitchell, H. E.: Tumors of the Larynx—A Review of 105 Cases, Laryngoscope 53:7 (July) 1943.

7. Hulse, W. F.: Hoarseness in Tuberculosis, Arch. Tuberc. 42:776-781, 1940.

8. Wells, Walter: The Significance of Hoarseness, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 49:99-112 (Mar.) 1940.

9. Carpenter, E. W., and Carpenter, W. M.: Chronic Hoarseness, South Carolina M. Assn. J. 33:281-310 (Dec.) 1937.

10. Clerf, L. H.: Treatment of Simple Chronic Laryngitis, Arch. Otolaryng. 27:261 (Mar.) 1938.

11. Orton, H. B.: The Significance of Hoarseness, Trans. Am. Laryn., Rhino., and Otol. Soc., 1942.

12. Zinn, W. F.: Laryngectomy; One Stage, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 47:453 (June) 1938.

XIII

SOME LESIONS ASSOCIATED WITH FISTULA OF THE FRONTAL SINUS

HAROLD L. HICKEY, M.D.

DENVER, COLO.

Fistula of the frontal sinus is an occurrence which can be classified both as a subjective symptom and an objective sign. It is the result of certain pathological changes produced by a variety of disease entities and for that reason it is given per se very little consideration in most of the standard texts of otolaryngology. However, the study over a period of years of a group of cases in which external frontal sinus fistula was the predominant finding common to all, in spite of various etiologies, prompted the writer to investigate the pertinent literature and to report some illustrative cases. In this article only those lesions will be considered which produce external fistula by spontaneous perforation through the anterior wall or floor of the frontal sinus. It is recognized that necrosis of the orbital plate internally or of the posterior wall may occur, with resultant orbital cellulitis or abscess in the one case and intracranial extension in the other, but such complications while they might be construed as fistulous are not visible externally and are therefore not in the scope of this discussion. Also, fistula of the frontal sinus acquired by reason of gun shot, fracture or other injuries as well as the result of previous surgery of the sinuses with incomplete healing is not considered in the limited field of this paper.

In the production of fistula of the frontal sinus the important factors are: (1) increase in intrasinus pressure incident to an expanding lesion with closure of the nasofrontal duct by swelling or tissue mass and (2) pressure necrosis and perforation of bone, subcutaneous tissues and skin to form the fistula. These changes may be produced incident to infection by acute or chronic frontal sinusitis or osteomyelitis of the frontal bone; by infectious granulomatous processes in the case of tuberculosis or syphilis; and independent of infection by mucocele or such neoplasms as chondroma, osteoma, or malignant growths.

Mosher[1] in 1904 gave the anatomical description on which may be predicated the location of frontal sinus fistula. He found the

average thickness of the anterior wall of the frontal sinus to be 4 mm., although it may be as little as 1 to 2 mm. or as great as 7 to 12 mm. Of the posterior wall the average thickness is 1 mm. but the bone is very compact. The orbital part of the floor is paper thin and represents a continuation of the os planum anteriorly and superiorly. "It offers but little resistance to pus." Thus it appears that fistula should occur more frequently through the floor, less frequently through the anterior wall. This is borne out by clinical observation.

Skillern[2] stated that fistula formation in suppurative sinusitis is due to perforation of the wall, which process has its inception during an acute exacerbation of a chronic inflammation. A small area of sinus mucosa, from pressure due to occlusion of the drainage passages, becomes necrotic with thrombophlebitis of the small veins which penetrate the bone. Infection is thus carried to the external periosteum of the sinus wall setting up an external purulent periostitis. The bone thus losing its nourishment softens and breaks down in this circumscribed area. In the case of mucocele or tumor, progressive bulging of the sinus wall causes pressure atrophy, thinning of the wall and finally perforation.

Hajek[3] gave a similar picture of the pathology and emphasis was placed on the role of the occluded or incompletely draining nasofrontal duct in the production of bone necrosis and fistula formation; he stated that the common site is the orbital roof behind the supraorbital notch. He offered as a possible explanation "the anatomic fact that in individual cases congenital defects exist in the orbital wall of the frontal sinus." The orbital process together with fistula formation may progress without pain in spite of extensive disease of the frontal sinus. This was a noteworthy finding in one of the cases reported below.

Acute frontal sinusitis as a cause of external fistula does not receive frequent consideration by authors of textbooks and no reported cases were found in the literature. However, Lederer[4] mentioned it as a cause of fistulous tracts and stated that they "usually occur through the anterior wall of the sinus or at the floor, opening above the inner canthus."

Lillie and Anderson[5] in a study of 60 cases of suppurative frontal sinusitis reported 37 with external fistula; of these, in 19 the opening was in the outer table, in 7 the fistula was noted in the orbit but was still external, and in the others fistula presented in the scar of previous surgery. The last group is not germane to the present discussion. It is thus evident that chronic suppurative disease of the frontal sinus

is a not uncommon cause of external fistula of the anterior wall or floor (orbital plate) of the sinus.

Shambaugh[6] reported a case of frontal sinusitis and ethmoiditis with external fistula one-fourth inch above the inner canthus of the eye. Fistula developed after six weeks of headache and an accidental kick from a child's shoe. Removal of the middle turbinate, exenteration of the ethmoids and exposure of the nasofrontal duct resulted in prompt closure of the fistula. X-ray examination showed the frontal sinuses to be small and the ethmoid and antrum involved on the same side as the fistula. Dr. J. C. Beck in discussion thought the case was due to ethmoiditis rather than to frontal sinusitis.

Osteomyelitis originating in the frontal sinus and extending to the frontal bone, orbit and intracranial structures does not usually produce fistula of the frontal sinus as the invasion is so rapid and fulminating by thrombophlebitis of the diploic venules that there is not sufficient time for pressure necrosis and fistulization to occur, except in low-grade localized infections. Thus Mosher and Judd[7] in their masterly description of osteomyelitis of the frontal bone did not report fistula formation in any of their cases. However, Weiss[8] reported a case in which localized osteomyelitis produced necrosis of the anterior wall and the floor of the frontal sinus; rupture of the abscess externally was impending. Extreme illness of the patient and numerous complications demanded surgery before actual fistulization took place.

Davis[9] described a case of osteomyelitis with fistula, orbital cellulitis and abscess, and diplopia complicating pansinusitis. It is not entirely clear by what process the fistulous tract originated or where it extended; it was successfully treated by a modified Killian operation supplemented by sulfathiazole therapy.

At the time Lockard[10] published his work on tuberculosis of the nose and throat in 1909, in a survey of 7645 tuberculous patients including 904 of his own there were only 38 cases of nasal tuberculosis; of these only 4 had frontal sinus involvement.

Lederer and Livingston[11] in an article on tuberculosis of the nasal accessory sinuses quoted Levy as authority for "the case of a child of two years who presented an abscess below the left eye and a fistula above the right eye. Death followed hemorrhage through the mouth and nose. At necropsy the body of the sphenoid was found destroyed as well as caseous masses in the frontal, ethmoid and maxillary sinuses". The clinical picture, although not stated to have been verified micro-

scopically, was obviously that of tuberculosis as the cause of the frontal sinus fistula. In Vohsen's case mentioned in the same article "a fistula in the forehead led to bone necrosis and caseous masses in the frontal sinuses."

Berruecos[12] in 1934 reported the case of a male, aged 53, with symptoms of cephalgia, left nasal obstruction, and serosanguineous odorous discharge. There were multiple nasal polypi in the left fossa, one extending to the outlet of the naris. Large polypi were removed via the nasopharynx and nose. They recurred 16 months later and a gradually enlarging tumefaction appeared over the frontal bone. Pus began to appear in the nose at a point away from the normal frontal outlet and pressure externally increased the flow through a probable fistula. In spite of negative serology for syphilis the condition subsided rapidly under vigorous antisyphilitic treatment and was considered to be a gumma of the frontal sinus with necrosis of the bony wall.

McLeod and Lux[13] cited a case of cicatricial ectropion as a result of mucocele of the frontal sinus which had originated 12 years previously. Swelling of the upper lid due to the frontal mucocele had been incised and drained one year after onset, after which it closed only to reopen spontaneously; the chronic irritation of the draining fistula stimulated scar formation, contraction and eversion of the eyelid into the border of the opening. This case simulates one of those described hereafter in this series and is the only one of its kind noted in the literature, yet this fistula was not entirely spontaneous in its origin.

Chondroma of the ethmoid which subsequently invaded the frontal sinus and the orbit, with resultant orbital abscess and external fistula, has been reported by the writer[14] in a previous article.

Primary osteoma of the frontal sinus existing concomitantly with mucocele and producing a fronto-orbital fistula was reported by Patriarcha and Rossello.[15] In the majority of cases, however, osteoma of the frontal sinus does not produce fistula and it is often discovered unexpectedly on x-ray examination of the sinuses.

Primary malignancy of the frontal sinus is relatively rare as attested by Schall,[16] Barnes[17] and others. The latter stated: "The frontal is rarely the seat of a primary growth. . . . Primary tumors of the frontal may be mistaken for chronic frontal sinusitis, and the correct diagnosis made only after the anterior plate is absorbed." That

it may be the source of fistula of the frontal sinus is shown by two of the cases here reported.

Fistula of the frontal sinus may not be as uncommon as a survey of the available literature might lead one to believe. But in view of the relatively small number of cases reported as such, it seems worth while to record the following interesting and somewhat unusual cases:

REPORT OF CASES

CASE 1. Mrs. N. P., aged 58, negress, was admitted to Colorado General Hospital November 13, 1940. Her complaints were pain and soreness in the supraorbital region, and a lump on the forehead present for four months, gradually increasing in size. The swelling began about two weeks following removal of nasal polypi, so extensive that a photograph was made by her physician (Fig. 1). No pathologic examination was made at that time but section of residual polyp tissue later revealed "benign edematous polyp" (Fig. 4). The past and family history were unimportant, she had two children living and well, no miscarriages. There was no family history of carcinoma; she was not subject to frequent colds; no history of epistaxis was elicited.

Physical examination revealed no essential findings (other than blood pressure 190/80) except in the head region. A firm nonmovable swelling about 3 cm. in diameter was seen in the frontal region, slightly to the left of the midline; the resistance of the area was celluloid-like and suggestive of thinned out bone. A 5 mm. linear fistulous opening above the left brow exuded some thin discharge (Fig. 2). The nose was free of polypi, a small amount of mucopus was seen beneath the left middle turbinate. There was no diplopia; bilateral arcus senilis was present.

Roentgenograms of the sinuses showed the outline of frontal sinuses indistinct; in lateral view they appear shallow; there is a bony defect near the upper border of the left frontal sinus possibly due to osteomyelitis. The blood count and urinalysis were normal. Routine Wassermann and Eagle tests were negative; a provocative Wassermann test was also negative.

Tentative, preoperative diagnoses were mucocele, pyocele or gumma of the left frontal sinus with fistula. Curiously enough the diagnosis of malignancy did not occur to any of the several staff members who saw this patient. There was evidence of vascular hypertension.

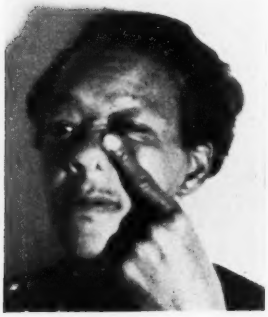


Fig. 1. Case 1.—Nasal polypi visible in left external naris.

On November 23, 1940, under general anesthesia I performed an external sinusotomy. A curvilinear incision was made to expose the left frontal sinus and on elevating the soft tissues there was a bony defect 1 x 1.5 cm. in diameter in the anterior wall of the frontal sinus directly beneath the small external fistula. Tissue appearing clinically malignant protruded through the opening and a frozen section examined during surgery revealed squamous cell carcinoma (Fig. 5). (Subsequent study of paraffin sections verified the frozen section diagnosis). On this information the incision was extended to expose both frontal sinuses which were widely opened down to the nasal bridge; malignant tissue had destroyed the interfrontal septum and large masses were removed from both frontal sinuses. The posterior wall of the left frontal sinus seemed less resistant than normal but no perforation was noted. The left ethmoid was exenterated and the rim of the left orbit, clinically malignant, was removed. Since the object of surgery was removal of carcinoma rather than to conserve ocular muscle control, diplopia was to be expected. However, the patient exhibited no diplopia at any time. A large intranasal opening was packed with oiled gauze and skin closure was made with dermal sutures, with a small Penrose drain at each external angle of the wound.

The patient exhibited comparatively little postoperative reaction and the wound healed well without appreciable deformity. Roentgen therapy was begun five days after the operation, and during a period of 21 days she received a total of 4600 r units. The resulting pigmentation, rather unusual in a negro patient, is still visible

Fig. 2. Case 1.—(a) Frontal swelling showing small fistula. (b) Lateral view of frontal swelling.

Fig. 3. Case 1.—As seen 30 months following surgery and irradiation. a. Lateral view shows lack of deformity. b. Frontal view shows pigmentation.

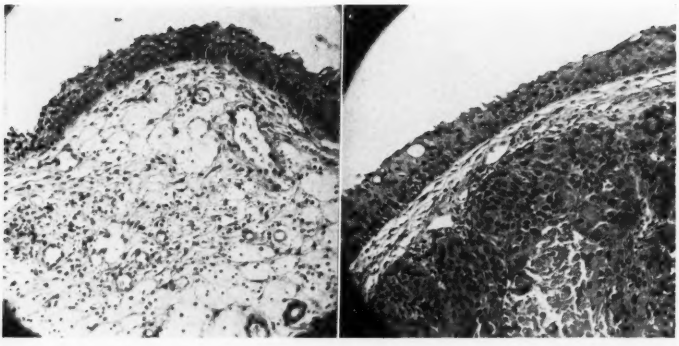


Fig. 4. Case 1.—Photomicrograph of nasal polyp.

Fig. 5. Case 1.—Photomicrograph showing squamous cell carcinoma.

June 18, 1943, some 30 months following treatment (Fig. 3). Frequent observation for a period exceeding four years has shown no evidence of recurrence of the lesion.

CASE 2. Mrs. F. A. N., aged 52, was first seen February 24, 1931, complaining of nasal obstruction and discharge without headache, irritation of both eyes especially the left. Nasal polypi had been first removed 27 years previously and on one or two occasions since that time but not for several years. She did not have frequent colds and aside from having always been somewhat underweight she enjoyed good health.

Physical examination revealed multiple nasal polypi in each fossa, a small amount of mucopurulent discharge in each middle meatus; the septum was irregularly deviated but not obstructive, the tonsils were adherent, slight discharge was seen in the nasopharynx. The ears were normal, the mouth was edentulous. There was a small fistula in the floor of the left frontal sinus above the inner canthus of the left eye and from this opening some thin discharge emanated. The patient did not think this important enough to list it in her complaints; on further questioning she thought the fistula had occurred about a year previously. The balance of the physical examination was essentially negative.

Roentgenograms of the accessory sinuses showed the right frontal sinus slightly larger than the left and clear; the left frontal slightly hazy inferiorly; and some loss of continuity of the adjacent orbital

Fig. 6. Case 2.—Appearance of sinuses in 1931.

rim. The interfrontal septum is not sharply defined and the usual lobulated appearance of the frontal sinuses has been replaced by a smooth rounded appearance. The right ethmoid is clear, the left is hazy; both antra are narrow and somewhat more dense than normal; the sphenoids are clear (Fig. 6).

A diagnosis of recurrent nasal polypi, chronic sinusitis and possible mucocele with fistula was made and both intranasal and external operations were advised.

On March 9, 1931, under cocaine and adrenalin anesthesia, preceded by sodium amytal, I removed multiple nasal polypi, opened and curetted both anterior ethmoids and exposed both nasofrontal ducts which could be easily probed; no characteristic discharge appeared at the time of intranasal surgery. Light oiled gauze nasal packing was removed on the following day. On May 30, 1931, the intranasal healing was satisfactory but the fistula continued to drain. External operation was advised but was declined by the patient as she felt well and had an adequate airway.

The patient was not seen again until some nine years later on February 19, 1940, when she was referred back by her oculist whom she had consulted because of failing vision in the left eye. At this time she presented a marked ectropion due to contraction of the upper lid into the fistulous opening in the floor of the left frontal sinus (Fig. 7a). The exposed cornea had been "protected" by the patient each

Fig. 7. Case 2.—a. Ectropion caused by fistula; note inability to close left eye.

Fig. 7. Case 2.—b. Appearance following plastic repair of fistula and etropion.

night with a silk scarf. The foreign body contact had caused corneal abrasion followed by scar and decreased vision. The patient stated that in the interim she had experienced no headache and was not disturbed in any way except by the annoyance of drainage from the fistula and inability to close the left eye.

On examination of the nose, no polypi were seen; considerable purulent discharge was noted in the right fossa, none in the left. On palpating the right middle meatus with a curved applicator, very slight resistance gave way and allowed the applicator to pass suddenly upward and backward some 8 or 10 cm. into a large cavity. Immediately there was a gush of thick foul mucopus which was aspirated by cannula suction in the amount of approximately 3 oz. The left nasofrontal duct was not patent.

Roentgenograms of the sinuses at this time exhibited a marked contrast to the former films: The cavity of both frontal sinuses is markedly enlarged, especially the right which extends almost to the lateral border of the orbit; there is evidence of absorption of the interfrontal septum, of the anterior and posterior tables of the right frontal sinus in its inferior portion and the upper third of the right orbit. On lateral view there appears a large cavity which may be in the anterior cranial fossa. The borders of the sinuses are surrounded

Fig. 8. Case 2.—Appearance of sinuses in 1940. Note large eroded cavity, also area of increased density surrounding left frontal sinus.

by a reactionary area of increased density suggestive of pressure necrosis. There is no definite evidence of osteomyelitis (Fig. 8).

From the clinical and x-ray evidence it appeared that an expanding lesion of the frontal sinuses, probably a mucopyocele, had eroded and largely destroyed the interfrontal septum, a portion of the right orbit and the posterior wall of the right frontal sinus, forming a large cavity containing the viscid secretion liberated on examination.

On March 7, 1940, under local anesthesia the region of the right ethmoid and nasofrontal duct was enlarged and explored releasing additional foul purulent drainage. On the following day the patient said she felt better than for the past six months. She was observed frequently and treated locally to keep the nasofrontal duct open by passing a No. 13F. nasal sound; the drainage gradually decreased to a minimal amount but the fistula from the left frontal continued to drain.

On July 9, 1940, the left nasofrontal duct was enlarged liberating considerable discharge, and nasal sounds passed into both ducts simultaneously could be felt in contact in the combined frontal cavity. On the following day the frontal fistula closed and showed no drainage thereafter.

There being no unfavorable reaction to the intranasal procedures and because the patient refused surgery by the external route, on Au-

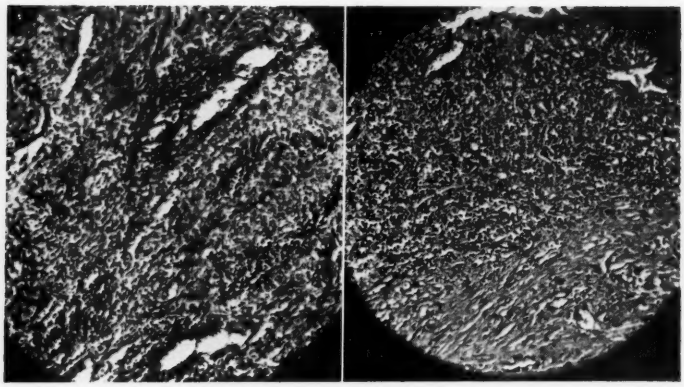


Fig. 9. Case 3.—Photomicrograph of tissue removed at first operation, showing squamous cell carcinoma.

Fig. 10. Case 3.—Photomicrograph of tissue removed at final operation, showing chronic inflammation and fibrosis.

gust 23, 1940, she was admitted to St. Luke's Hospital for plastic repair of the fistula and ectropion by Dr. D. W. Macomber. Scar tissue was removed, so as to free the upper lid from its attachment to the orbital roof and floor of the frontal sinus; no open fistulous tract was seen. A two-thirds thickness skin graft was laid in the defect and held in place by sutures of black silk over a mold of dental compound. On the tenth day the sutures and mold were removed and the graft was in excellent condition.

By the plastic procedure lid function was restored, the exposed cornea was normally covered and the palpebral fissure which had been distorted was rendered about equal to its fellow on the right side. The appearance in an "intermediate" stage of healing with the graft still discernible may be seen in Fig. 7b. A further improvement in appearance and function was later evident without additional surgery.

As it sometimes happens in the case of indigent or semi-indigent patients, a final photograph for record in this case was not obtainable. The nasofrontal ducts are known to have remained patent up to August 3, 1942, when the patient was last seen. A recent attempt to persuade the patient to allow further x-ray studies to be made with the aid of an opaque media was stubbornly refused, but a telephone

Fig. 11. Case 3.—Appearance following operation on left frontal sinus.

report is to the effect that she is apparently in good health almost five years following discovery of the large eroded cavity. It is still felt that the safer and certainly more informative method of approach would have been by the external surgical route. Such are the vagaries of the human race.

CASE 3. J. C. D., aged 51, white male, was admitted to the Colorado General Hospital September 28, 1942, because of a draining fistula in the floor of the left frontal sinus. He brought with him a statement from a physician in a distant town stating that he had operated upon the left frontal sinus August 26, 1942, and that the subsequent course of the case had been unsatisfactory. A report from a reputable pathologist gave the diagnosis of "squamous cell carcinoma of the left frontal sinus". It was not definitely determined whether the fistula preceded the surgery.

Past history revealed symptoms of chronic sinusitis for several years, seasonal hay fever in August and September of each year and occasional attacks of asthma. A diagnosis of Hodgkin's disease, made by biopsy of a cervical gland about 20 years ago, had been treated by several series of x-ray treatments, the last in 1934, since which time there had been no glandular enlargement.

Physical examination revealed a fistulous opening in the floor of the left frontal sinus medial to and behind the supraorbital notch; pus drained from the opening. Slight swelling of the left periocular tis-

sues and some conjunctivitis of the left eye were noted. There was a moderate amount of purulent discharge in the left nasal fossa, the left nasofrontal duct could not be probed; the septum was deflected to the right, the tonsils were atrophic, the mouth edentulous; the ears were normal. A small left cervical gland was palpable, otherwise there was no adenopathy or evidence of Hodgkin's disease. The remainder of the physical findings were normal.

Roentgenograms of the sinuses showed extensive irregularity and enlargement of the frontal sinuses predominantly on the left; marked clouding of the left antrum and the left ethmoid cells. Blood count and urinalysis were normal, Wassermann and Eagle tests were negative.

Preoperative diagnosis was chronic left frontal sinusitis with fistula, possible malignancy.

On October 3, 1942, under general anesthesia I performed a modified Lothrop external sinusotomy as follows: A curvilinear incision was made through the left brow downward to the nasal bone, the soft tissues were elevated upward leaving the orbital tissues and fistulous opening undisturbed. The frontal sinus was entered through the anterior plate and the opening enlarged to 2.5 x 1.5 cm. The lining mucosa was thickened and bathed in pus; the internal end of the fistula was seen but not disturbed. The mucous membrane was removed as completely as possible from an enormous sinus; the frontal duct was located and enlarged by rasping from above downward to a diameter of about 7 mm. The periosteum was closed with catgut, the skin wound and fistula were closed with black silk. A Penrose drain was placed from the sinus to the external angle of the wound, and a small iodoform oiled gauze pack was placed in the nose. All bone and soft tissue removed at operation was sent to the laboratory for microscopic examination.

Aside from a rise in temperature to 101° F. on the first postoperative day, the course was uneventful. Careful examination of tissue specimens failed to reveal any malignancy; only chronic inflammation with granulation and some fibrosis was reported (Fig. 10). A review of the slide of tissue previously removed leaves no doubt as to the malignant character of the original lesion (Fig. 9). It is possible that an early, well-localized, malignant focus was removed in its entirety. This patient has been followed with frequent examinations for more than two years and there is no evidence of malignancy. The nasofrontal duct remains patent, the wound is well healed and minimal deformity is apparent (Fig. 11).

SUMMARY

Some of the varied lesions associated with external fistula of the frontal sinus have been discussed together with the etiology and the mechanism of fistula production. The pertinent literature has been reviewed and illustrative cases are reported.

934 REPUBLIC BUILDING.

REFERENCES

1. Mosher, H. P.: Applied Anatomy of the Frontal Sinuses, Laryngoscope 14:830-855 (Nov.) 1904.

2. Skillern, R. H.: Accessory Sinuses of the Nose, Ed. 3, Philadelphia and London, J. B. Lippincott Co., 1920, p. 251-252.

3. Hajek, M.: Diseases of the Nasal Accessory Sinuses, Ed. 5, Vol. II, St. Louis, C. V. Mosby Co., 1926, p. 322-325, 342-343.

4. Lederer, F. L.: Diseases of the Ear, Nose and Throat, Ed. 2, Rev., Philadelphia, F. A. Davis Co., 1939, p. 399-400.

5. Lillie, H. I., and Anderson, C. M.: The Two Stage Operation for Suppurative Frontal Sinusitis with External Manifestations, Arch. Otolaryng. 5:154-161 (Feb.) 1927.

6. Shambaugh, G. E.: Proc. Chicago Laryng. and Otol. Soc. in ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 23:937 (Dec.) 1914.

7. Mosher, H. P., and Judd, D. K.: An Analysis of Seven Cases of Osteomyelitis of the Frontal Bone Complicating Frontal Sinusitis, Trans. Am. Laryng. Rhin. and Otol. Soc., 1933, p. 289-242.

8. Weiss, J. A.: Circumscribed Perforative Osteomyelitis of the Frontal Sinus, Illinois M. J. 74:168-170 (Aug.) 1938.

9. Davis, J. S.: Osteomyelitis with Fistula, Orbital Cellulitis and Abscess, and Diplopia Complicating Pansinusitis, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 52:906-911 (Dec.) 1943.

10. Lockard, L. B.: Tuberculosis of the Nose & Throat, St. Louis, C. V. Mosby Co., 1909, p. 269.

11. Lederer, F. L., and Livingston, G. S.: Tuberculosis of the Nasal Accessory Sinuses, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 37:1176 (Dec.) 1928.

12. Berruecos, P. T.: Un caso de Fronto-Etmoiditis Crónica Fistulizada por la Región Frontal, An. Soc. mex. de oftal. y oto-rino-laring. 11:121-126 (Oct.-Dec.) 1936.

13. McLeod, J., and Lux, P.: Cicatricial Ectropion as a Result of Mucocele of the Frontal Sinus, Arch. Ophth. 15:994-997 (June) 1936.

14. Hickey, H. L.: Chondroma of the Ethmoid, Arch. Otolaryng. 31:645-652 (Apr.) 1940.

15. Patriarcha, A. P., and Rosello, G.: Osteoma primativo del seno frontale concomitante a mucocele fistolizzato nell' orbita in correspondenza dell' angolo supero-esterno. Riv. oto-neuro-oftal. 11:153-161 (Mar.-Apr.) 1934.

16. Schall, L. A.: Personal conversation with the author.

17. Barnes, H. A.: Neoplasms of Nasal Accessory Sinuses, in The Nose, Throat and Ear and Their Diseases, Edited by Jackson and Coates, Philadelphia and London, W. B. Saunders Co., 1929, p. 169-170.

XIV

LARYNGEAL STROBOSCOPY

H. B. PERLMAN, M.D.

CHICAGO, ILL.

Information obtained from recent ultra high speed moving pictures of the vocal cords during phonation has re-emphasized to the laryngologist the shortcoming of the ordinary mirror examination of the larynx. This shortcoming has long been recognized by the students of speech and led early to their use of the stroboscope in studying the action of the vocal cords during phonation. The stroboscope was first used by Oertel[1] in 1878 for this purpose. He and a few of his contemporaries[2-4] wrote rather extensively about their observations some fifty years ago. Their laboratory equipment did not lend itself to widespread use and the method failed to receive widespread and enduring acceptance. Only sporadic accounts of laryngeal stroboscopy have appeared from that time until recent years. In the past decade, students of speech[5-10] have renewed their interest in this subject and have used the gas-filled glow lamp, electrically discharged, as an improved source of interrupted light. The clinical laryngologist has indicated little interest in this work at any time. This may be due in part to the fact that the equipment designed for stroboscopic examination did not lend itself readily to clinical use.

It is apparent to anyone called upon to diagnose and to treat laryngeal disease and to demonstrate normal and pathological vocal cord movement to students that it would be distinctly useful to be able to visualize readily the movement of the vocal cords during phonation. The gross movement of the cords during abduction and adduction ordinarily observed represents only the elementary function of the larynx. While high speed moving pictures of the diseased larynx during phonation would be the ideal method of examination, it is not practical. The visual picture obtained with the stroboscope is the best substitute. The use of an electrically driven gas tube as a source of light gives a more accurate stroboscopic image than that obtained from a light source mechanically interrupted but is less readily

---

From the Division of Otolaryngology of the University of Chicago.

This work was done in part under a grant from The Douglas Smith Foundation for Medical Research of The University of Chicago.

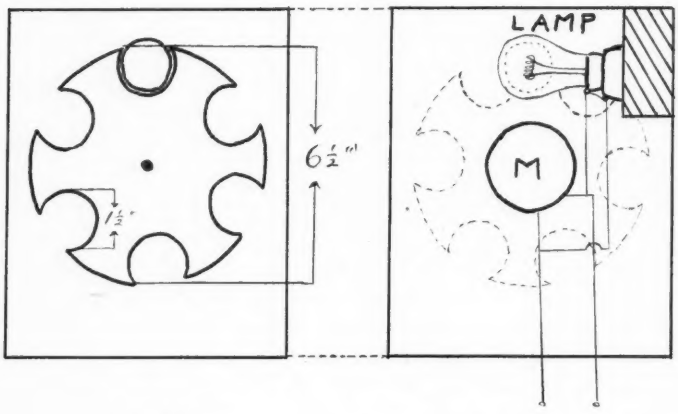


Fig. 1.—Diagram of stroboscope. Numbers indicate the diameter of the disc and of the disc perforations in inches. Rear view indicates the arrangement of the motor (M) and light within the box.

adapted to clinical use. Furthermore, in studying gross disease of the larynx it is not essential to be able to visualize cord movement of widely varying frequency. Observation of the cord movement for a single pitch of the voice is adequate.

A simple, small, portable stroboscope was constructed (Fig. 1) that could be used as a light source for the head mirror. It consists of a small box containing an 1800 cycle a.c. motor and an ordinary frosted light bulb with circular window. The box is perforated for the light and for the shaft of the motor. To the shaft is attached an easily demountable aluminum disc 6½ inches in diameter. Six holes placed at the circumference of the disc are equal in size to the hole in the box and to the window in the frosted light bulb. The circuit is so arranged that the motor and the light are started by a single control. The stroboscope is mounted in the same position as the ordinary light for indirect mirror examination of the patient (Fig. 2). With the disc turning 30 times a second and with each turn allowing six interruptions of the light source, 180 flashes of light per second are obtained.

The success of this method of laryngeal examination is based upon the fact that the subject can control the frequency of his vocal cord vibration.

In order that the subject may easily approximate his vocal cord movement to the frequency of light interruptions and thus permit the stroboscopic effect, the desired pitch is determined by a tuning fork. A weighted 128 cycle steel tuning fork is used for this purpose. The weights are pushed down equally on the prongs of the fork until the vibration of the prongs of the fork can be seen in the stroboscopic light. The fork is now sounded at the subject's ear and the subject asked to pitch his voice accordingly. Another satisfactory and even more simple way is for the examiner to make the properly pitched sound and ask the subject to simulate it. The average patient has no difficulty in setting the pitch of his voice in this way even though the tongue is firmly held and the mirror is in place.

The examiner now sees the undulations of the cord during sound production. A loud voice produces wider and more easily seen excursions of the cord than a soft voice. The speed of the to and fro movement seen is dependent on the difference between the frequency of vocal cord vibration and the frequency of light flashes. If no difference prevails no movement would be seen. This condition rarely prevails and only for a very short time. If the difference is one cycle per second the examiner sees one vibration of the cords per second. If the difference is five cycles per second the cords will appear to the examiner to be vibrating five times a second. The high degree of cortical control over the frequency of the vocal cord vibration during phonation is readily apparent by the ability of the subject to change the stroboscopic picture at will from one of slow to one of rapid movement or vice versa.

The adult male can easily pitch his voice so that his cords move about 180 times per second. The adult female can also pitch her voice to the same frequency although a higher fundamental frequency of cord movement is more easily produced. This is in the neighborhood of 240 cycles per second. For stroboscopic examination of the female larynx the same six-holed disc is satisfactory. The use of the same disc for stroboscopic examination of the male and female larynx simplifies the procedure. However, an eight-holed disc may be substituted readily for examination of the female larynx. The value of this method for demonstrating to the student normal laryngeal function during phonation is apparent.

A few illustrative cases serve to indicate the value to the laryngologist of this method of examining the diseased larynx.

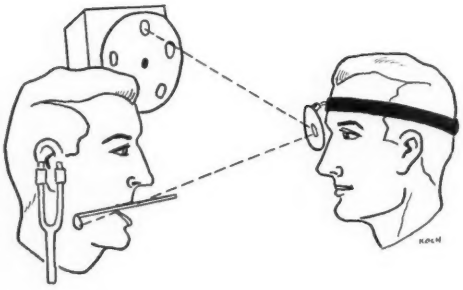


Fig. 2.—Schematic drawing showing the position of the stroboscope with respect to the patient and to the examiner. A tuning fork is placed at the ear of the subject to indicate the manner of obtaining the proper frequency of cord vibration.

REPORT OF CASES

CASE 1. Mr. P., aged 61, had had hoarseness of nine months' duration when examined on May 26, 1943. Both cords were red and thick. The left cord was irregular throughout, but definite though reduced movement in adduction and abduction was visible. Stroboscopic examination revealed that the right cord could be made to oscillate near the frequency of the light flashes. The left cord at the same time appeared still. Biopsy of the left cord showed an immature carcinoma. Six months after roentgen therapy his voice was much improved. The left cord was now smooth, moved well grossly and could be made to oscillate at the frequency of the stroboscope. However, the frequency of this oscillation could not be well maintained.

Comment. The normal stroboscopic picture of the cords is one of identical, symmetrical oscillation of opposing cords. This patient in attempting to pitch his voice to the frequency of the tuning fork could accurately control the vibration of the right cord but the left cord did not respond. The left cord could have been vibrating at a higher or lower frequency during this time. If the frequency of vibration was more than about ten cycles different from the frequency of the stroboscope, the cord would appear to remain still. The hoarseness was due to his inability in phonation to bring his left vocal cord into movement identical with that of the opposing right cord. The carcinoma had not infiltrated the cord or the crico-arytenoid articulation to a degree sufficient to prevent some abduction and adduction on deep inspiration and initial phonation. With dissolution of the carcinoma, control over cord movement for phonation was more nearly normal.

CASE 2. Mr. K., aged 58, had had hoarseness for five months when examined June 22, 1943. The left cord was thickened throughout and irregular and did not move on deep inspiration or upon initiating phonation. However, careful observation revealed that the arytenoid did move in abduction and adduction. Stroboscopic examination revealed the patient's ability to set his right cord into vibration at the desired frequency in spite of the fact that the left cord appeared still. The cord itself was fixed while the crico-arytenoid joint was still uninvolved. This was verified at operation. The carcinomatous cord and a part of the vocal process of the arytenoid was removed through a laryngofissure. After the cord was resected and while the larynx was open, the arytenoid could be seen to move freely and violently in abduction and adduction.

CASE 3. Mr. N., aged 50, had his right cord resected for carcinoma in 1941. When examined two years later he had a husky voice with no signs of local recurrence. The opposite cord moved well and was grossly normal. The site of operation was occupied by fixed connective tissue. During phonation the movement of the remaining cord was difficult to visualize because of the approximation of the false cords. With the stroboscope the tissue of the false cords was seen to vibrate at the frequency of the control fork. The subject had cortical control over the frequency of vibration of the laryngeal musculature entering into the false cord. The fundamental frequency of the voice was probably produced by these folds. The contour, method of apposition, and irregularity of this tissue did not permit reproduction of a fundamental sound like that obtained from the normally vibrating true cords, hence the difference in quality of the voice.

CASE 4. Mr. W., aged 55, has been hoarse for two years. There was extensive thickening and irregularity of both cords with many white plaques, and hyperplastic tissue below the anterior commissure. His voice was very hoarse; but definite, free abduction and adduction of the cords were observed. Stroboscopic examination showed no ability to control the frequency of vibration of the cords to approach that of the stroboscope. No cord vibration during phonation could be visualized. The diagnosis is still obscure after three biopsies but suggests leukoplakia. The patient's glottic chink is so distorted and the cords so diseased that accurate control of their vibration frequency in phonation is not possible.

CASE 5. Mr. M., aged 43, first noted difficulty with loud, high-pitched singing voice in 1939 after an intensive period of singing practice. A nodule was then found on the free edge of the left cord near the posterior commissure and a contact ulcer on the opposite cord. The ulcer was cauterized once and the nodule removed twice. The sections were negative for tuberculosis. No lasting improvement was obtained and the first examination in the department of otolaryngology on May 7, 1943, revealed the nodule and contact ulcer. Throughout this illness the patient has had a normal conversational voice. Stroboscopic examination revealed that during voice production of moderate intensity the involved segments of vocal cord did not vibrate—but appeared to remain at rest in the adducted position. When he increased the intensity of his voice, the entire cord was set into vibration, including the diseased portions. Upon this information, he was not placed on complete voice rest but was allowed to use a moderate, relaxed conversational voice. Examination five months later revealed that the lesions had entirely disappeared and the cords were normal.

Comment: The ability to see the cords in vibration during phonation permitted an explanation of the clear conversational voice in the face of definite cord lesions and indicated the value of moderation in voice production to give rest to the lesions.

CASE 6. Mr. M., aged 40, had had the following complaints for six months when examined on August 20, 1943. "Slight huskiness of voice; voice tires easily; cannot produce a loud voice; cannot carry a tune." Examination revealed a pinhead-sized translucent mass on the free edge of the right vocal cord at the junction of the anterior and middle third. This mass prevents accurate apposition of the two vocal lips, so that during phonation an elliptical space is seen between the cords. Stroboscopic examination showed that the subject could control the frequency of cord vibration in phonation. The mass vibrated at the same frequency as the cord to which it is attached. Close apposition of the cord lips during any phase of vibration was not seen. Even when the voice intensity was increased, complete closure of the glottic chink during a vibration cycle did not occur.

Comment: Failure to close the glottic chink during some phase of the voiced oscillation accounts for the change in quality of the voice. The need for a great air flow to keep the separated cords vibrating during phonation results in easy tiring of the voice. Failure to get true apposition of the cords reduces the volume or intensity of the voice. In singing, the subject can apparently control the frequency of vocal cord vibration but this does not lead to a good musical tone. This is due to the interference by the lesion with the exact manner of apposition of the cords for interrupting the air flow in the production of a singing voice.

CASE 7. Mrs. S. was examined December 1, 1943, for hoarseness due to paralysis of the left vocal cord. The subject could approximate the cords at the beginning of phonation since the paralyzed cord was near the midline but she could not maintain symmetrical oscillation of the cords. The normal cord was seen with the stroboscope to oscillate constantly at the desired frequency (near 180 cycles per second). The paralyzed cord could be seen to oscillate for a few cycles only at the onset of each "voicing." Sustained oscillation of the paralyzed cord at the frequency of the normal cord could not be maintained.

Several patients with paralysis of the vocal cord were examined with the stroboscope. When the cords could be apposed, the involved cord could be made to oscillate at the desired frequency for only a few cycles. One patient, however, whose left cord failed to abduct on inspiration could maintain the cord in oscillation as well as the opposite cord. He had a good voice. A mediastinal mass affected the nerve fibers to the muscles producing abduction of the cord on inspiration but the nerve fibers controlling the muscles that determine the oscillating properties of the cord in phonation were spared.

CASE 8. Mr. R., aged 57, was examined December 10, 1943. He complained of hoarseness of two years' duration. Both true cords were irregular, red and thick, particularly the left anterior halves. Movement was present in both cords and the posterior segments could be widely abducted on deep inspiration. Stroboscopic examination revealed that only the right cord could be made to oscillate at the desired frequency. The left cord could not be made to oscillate at the same frequency as the right or at any frequency near that of the stroboscopic light interruptions. Biopsy of the left cord showed early, well-differentiated carcinoma. The infiltration

of the carcinoma into the vocal cord interferes with the accurate, delicate control by the laryngeal muscles over the physical factors of tension mass and resistance of the vibrating cord.

SUMMARY

1. The importance to the laryngologist of assessing vocal cord movement during phonation is pointed out.

2. A practical method for making these observations, using a simple stroboscope, is described.

3. Stroboscopic findings in several illustrative cases of laryngeal disease are presented and discussed.

950 East 59th Street.

REFERENCES

1. Oertel, M. J.: Ueber eine neue laryngostroboskopische Untersuchungsmethode, Centralbl. f.d. med. Wiss. 16:81, 1878.

2. Musehold, A.: Stroboskopische und photographische Studien über die Stellung der Stimmlippen im Brust-und Falsett-Register, Arch. f. Laryngologie 7:1, 1898.

3. Spiess, G.: Ein neues Laryngostroboskop, Arch. f. Laryngologie 7:148, 1898.

4. Koschlakoff, D.: Ueber die Schwingungstypen der Stimmbander, Pflüger's Arch. f.d. Ges. Physiol. 38:428, 1886.

5. Clary: La Phonoscope á Cordes Vibrantes, Revue Scientifique 70:461, 1932.

6. Pollak-Rudin, R., and Stein, L.: Ein neues handliches Laryngostroboskop, Wiener med. Wschr. 82:916, 1932.

7. Kallen, L. A., and Polin, H. S.: A Physiological Stroboscope, Science 80:592, 1934.

8. West, R.: A View of the Larynx Through a New Stroboscope. Quart. J. Speech 21:355, 1935.

9. Moore, P.: Vocal Fold Movement During Vocalization, Speech Monographs, Vol. 4, 1937.

10. Tiffin, J.: An Approach to the Analysis of the Vibration of the Vocal Cords, Quart. J. Speech, Vol. 24, Feb., 1938.

XV

PENICILLIN AND THE INFECTIONS OF THE EAR, NOSE AND THROAT

M. H. Mothersill, M.D.

Indianapolis, Ind.

Infections constitute a large and important group of the diseases that distress mankind. Seventy years ago the majority of physicians did not believe that infections were due to invasion of the body by micro-organisms. As soon as that concept was accepted, medicine began to progress with amazing rapidity.

Since that time many methods of preventing or treating infection have been introduced. Vaccines to stimulate the production of active immunity and antisera to provide passive immunity against certain organisms or their toxins have been used with success. Various chemicals have been used as antiseptics for the skin and mucous membranes and, as time has gone on, improved antiseptics with greater tissue compatibility have been developed.

As soon as infection was understood, investigators began to search for agents which could circulate safely in the blood and the various tissue fluids of the patient and could inhibit or destroy pathogenic organisms. There are now available quite a number of these substances. Some are called chemotherapeutic agents, some are classified as antibiotics, and some cannot be put in either class. All of them taken as a group are designated as systemic anti-infectives.[1]

Quinine, for example, is a systemic anti-infective because it can be absorbed safely into the circulation and can there destroy the parasite of malaria. It is not ordinarily called either an antibiotic or a chemotherapeutic agent.

The term "chemotherapeutic" is applied to systemic anti-infectives which are synthesized by the chemist. The creation of some of these drugs has been the result of patient, methodical research by chemists who built, so to speak, a molecule with high antispirochetal or antibacterial potentialities and low toxic potentialities so far as the

---

Presented before the Middle Section of the American Laryngological, Rhinological and Otological Society, Indianapolis, Ind., January 17, 1945.

host is concerned. Examples of this group are the arsenicals and the bismuth compounds used in the treatment of syphilis, and more recently the sulfonamides.

An antibiotic is defined at the present time as a metabolic product of one micro-organism which is capable of inhibiting or destroying some other micro-organism. In order that an antibiotic may also be a systemic anti-infective, it must be able to circulate safely in the body and inhibit a pathogen. Penicillin is at present the outstanding antibiotic.

The Sulfonamides. Sulfanilamide undoubtedly represented a great advance in systemic anti-infectives; but, too often, it caused cyanosis, acidosis, hemolytic anemia, and various signs of toxicity to the nervous system such as dizziness, incoordination, and even psychosis. Sulfathiazole, sulfadiazine and sulfamerazine do not cause cyanosis or acidosis, and are less likely to cause hemolytic anemia or central nervous system symptoms. Moreover, they are more effective than sulfanilamide for equal concentrations. However, due to their lower solubility, these drugs may obstruct the urinary tract with crystals. Their solubility in urine is decreased by acidity and increased by alkalinity. A patient with a high fever frequently has a low output of acid urine. If full doses of sulfadiazine, for example, are being given, the urine may become an almost saturated solution of the drug or its acetylated derivative. If then the temperature of the urine falls, its ability to hold the drug in solution is further decreased and precipitation of crystals is more likely to occur.

The way to avoid crystalluria is *not* to reduce the dose of the drug to inadequate levels, but rather to increase the intake of fluid and the pH of the urine. If the dose is inadequate, the infection is not only not cured but may be rendered incurable because of development of sulfonamide-fastness by the pathogenic invader.

The possibility of the patient becoming allergic to the sulfonamides has been a source of concern. Many physicians refuse to prescribe sulfonamides in mild infections, where they might otherwise be beneficial, lest the patient develop allergy to all sulfonamides and be unable to take them at a later date when they are needed desperately. The percentage of patients capable of developing such hypersensitivity is low but unpredictable.

Still another handicap of the sulfonamides is the fact that although much of their usefulness lies in their effectiveness against pus-forming organisms, yet their activity is reduced by the presence of pus.

So there grew up a desire for a new systemic anti-infective which would be more potent and less toxic than the sulfonamides; one that would be so soluble that it would not block the urinary tract with precipitated crystals; one that would be effective against bacteria that had developed sulfonamide-fastness; one that could be taken safely by a patient who has become allergic to sulfonamides; and one whose effectiveness is not impaired by pus. In many respects penicillin meets these requirements. It is more effective than the sulfonamides against most of the pyogenic cocci and in doses which are less toxic. Bacteria can become penicillin-fast if inadequate doses are given; but the low toxicity of penicillin makes the giving of adequate doses safer. Allergy to penicillin is possible, but it will probably be a rare coincidence in which a patient becomes allergic to both penicillin and the sulfonamides.* The activity of penicillin is not markedly reduced by pus, although it is better for mechanical reasons to remove pus.

Penicillin. Penicillin will not replace the sulfonamides completely. It is ineffective against gram-negative bacilli, and some of these are susceptible to sulfonamide therapy. In an infection due to an organism which is susceptible both to penicillin and the sulfonamides, there is reason to think that full doses of both agents will produce greater effect than either one alone. If this is substantiated, there will be cases in which both drugs are given simultaneously.

Penicillin is with a few exceptions more effective and more desirable than the sulfonamides for the treatment of infections due to: staphylococci, the clostridia that cause gas gangrene, hemolytic streptococci, anaerobic streptococci, pneumococci, meningococci, gonococci, and anthrax. It is effective also in syphilis and actinomycosis. In tetanus[2] and diphtheria it is not directly antitoxic, but it is antibacterial and can thereby prevent further production of toxin. Its use as a supplement to antitoxin therapy may prove very valuable in the treatment of these diseases.

Fleming's[3] discovery of penicillin arose from the fact that the mold, *Penicillium notatum,* became an accidental contaminant of one of his culture plates of staphylococci. The antibiotic substance produced by this mold had the ability to diffuse out into the medium and destroy staphylococci. Fleming noted that in a fairly wide zone around the colony of mold staphylococci began to undergo lysis. What had originally been a well-grown staphylococcal colony was now a faint shadow of its former self. Fleming transferred this mold to liquid media and it grew on the surface, but the liquid eventually was found to contain the antibiotic in solution in sufficient concen-

tration to inhibit completely the growth of staphylococci even when the medium was diluted 500 to 800 times. In 1932 Fleming[4] reported having applied filtrates containing this active principle to septic wounds with very good clinical results.

Chain, Florey, and associates, of Oxford University,[5, 6] developed methods of obtaining penicillin from Fleming's filtrate in the form of a dry powder which was more stable and contained the active principles in more nearly pure form.

The quantity of penicillin present in the powder was measured by its inhibitory effect upon staphylococci. A standard strain of staphylococcus was selected and a unit of penicillin was defined as that amount which, when dissolved in 50 cc. of meat extract broth, would be just sufficient to inhibit completely the growth of the test strain. Later the plate method of assay was used. A cylinder of a certain size was fitted accurately to the surface of nutrient agar. When the cylinder was filled with a solution containing one unit of penicillin per cc., the antibiotic diffused into the agar around the cylinder in sufficient strength to inhibit the growth of staphylococci in a zone whose width was 24 mm. The unit, so measured, was called the Oxford unit. In the future we will hear of the International unit. It is approximately equal to the Oxford unit but is defined as the specific penicillin activity contained in 0.6 microgram of the International Penicillin Standard. This standard was described at the International Conference on Penicillin last October as a specimen of the pure crystalline sodium salt of Penicillin II or G.

Recently chemists have succeeded in producing active crystalline forms from the powdered commercial penicillin that is now being supplied. They have identified not just one, but at least three different substances. American chemists have named these three substances Penicillin F, Penicillin G, and Penicillin X. British chemists designated the same three substances, respectively, I, II and III. These penicillins are qualitatively similar, though not necessarily identical, in their antibiotic action. Quantitatively they are different. In other words, they contain different numbers of units per milligram. Crystalline Sodium Penicillin G contains 1,666 units per mg. Production of crystalline penicillin for commercial use is not at present feasible, but in the laboratory it sheds considerable light on the chemical composition.

Studies of this crystalline penicillin suggest that the molecular weight is between 300 and 400, and that it is monobasic. The crystalline sodium salt is almost white and is rather stable.

First attempts in our own laboratories to produce penicillin resulted in a crude product containing 8 to 10 units per mg. A little arithmetic will show that this was almost 99.44 per cent impure. There was considerable reluctance to administer such a crude substance intramuscularly or intravenously, and so oral administration was tried. It was believed that the acid of the gastric juice should be neutralized before penicillin entered the stomach, and calcium carbonate was selected as the antacid. In four out of five cases of subacute bacterial endocarditis in which 5 to 8 grams of this crude material was given by mouth daily, a remission was observed shortly after the treatment was begun and a relapse followed cessation of penicillin dosage. The fifth case showed no response. Whether or not these remissions were coincidences or an evidence of penicillin activity, we are not in a position to say.

Free *et al.*[7] gave oral doses of 100,000 units of penicillin to human subjects and measured the excretion of this agent in the urine. When they also gave a dose of 10 gm. of sodium bicarbonate, the total excretion of penicillin in the urine varied from 1,950 to 12,700 units. When no alkali was given, the urinary excretion was 8,800 to 33,600 units. More study of the possibilities of oral administration of penicillin is in order.

Early in 1943, penicillin containing 250 and more units per mg. was being produced, and further clinical trial had to do with intramuscular, intravenous and local administration. As methods of preparation have improved, the purity of the product has steadily increased.

Soon after penicillin reaches the blood it disappears very rapidly. If a dose of 10,000 units is given intravenously, a more than adequate blood concentration for most infections is found immediately after the injection and a less than adequate concentration about 30 minutes later. When it is given intramuscularly, absorption requires some time and a moderate, but perhaps adequate, blood concentration may last for three hours.

In attempting to define adequate blood concentration, we must take into account the organism that we are trying to destroy. Some staphylococci are killed by a concentration of as little as 1/50 unit per cc., but other strains of staphylococci may require 2 units or more per cc. Aagesen[8] pointed out that an intramuscular injection of 25,000 units produces blood concentrations no higher than 1 unit per cc., and this only during the first hour. In view of the low tox-

icity of the present commercial form of penicillin, it will be far better to err on the side of excessive dosage as soon as adequate supplies of penicillin are available.

In otorhinolaryngology penicillin is used at times as a local, and at times as a systemic, anti-infective.

Swanson and Baker[9] gave penicillin intramuscularly in appropriate cases of acute otitis media with good results. In postoperative mastoidectomy wounds they administered it topically through a rubber tube. The wound was closed except for this tube and the tube was closed. At intervals of six hours or more the tube was opened for aspiration which was followed by instillation of fresh penicillin solution and then closure of the tube again. Local application was used in cases of chronic otitis media with sufficiently large perforation of the drum so that the solution of penicillin could enter the middle ear.

Putney[10] reported seven cases of osteomyelitis of the frontal or maxillary bone. He concluded that the optimum time for operative measures in acute spreading osteomyelitis is during the period when the infection has been checked by penicillin. Attempts to depend solely upon penicillin without the aid of surgery have been disappointing.

The status of penicillin in the treatment of sinusitis cannot yet be evaluated. It cannot, of course, be useful except as the sinusitis is caused or complicated by a penicillin-susceptible organism. Putney reported that acute infections of maxillary sinuses had been cured after several local applications of penicillin solution containing 250 units per cc. One case of chronic purulent infection of a maxillary sinus was described in which the cultures became negative but the discharge continued. Crowe[11] states that local application in the sinuses will give better results in acute than in chronic maxillary sinusitis. His explanation is that in the acute phase the bacteria are on the surface of the mucous membrane, but in the chronic stage they are deep in the submucosal tissue. To be effective, he holds that penicillin must be in direct contact with the infecting bacteria for several hours. In chronic infections, penicillin will perhaps be more effective if used to supplement surgical operations. Sale and Diamond[12] reported a case of long-standing purulent bilateral maxillary sinusitis with polyposis. The infecting organism was a staphylococcus. They introduced spinal puncture needles into each antrum and kept them in place for 24 hours. Every three hours 5 cc. of a solution of penicillin was introduced into each antrum, after which the stilet was

replaced. The patient was kept in an upright position. The results were good.

Efforts have been made to apply penicillin effectively to the general surface of the nasal and oral mucosa. Some British investigators in 1941[13] suggested the use of penicillin snuff composed of 1 part penicillin, 5 parts menthol, and 94 parts by weight of lycopodium. More recently another group of British investigators[14] have used lozenges of penicillin which are capable of liberating the drug in active form in the mouth. They suggest using such lozenges following surgery of the mouth or throat.

SUMMARY

1. As a local and systemic anti-infective, penicillin is usually more active against pyogenic cocci than the sulfonamides.

2. It is ineffective against gram-negative bacilli. Infections due to some of these are susceptible to sulfonamide therapy.

3. It appears to have value in the treatment of certain infections, such as syphilis, diphtheria and tetanus, in which the sulfonamides are useless.

4. Penicillin is low in toxicity. Allergy to penicillin can occur, but allergy to both penicillin and the sulfonamides is likely to be an unusual coincidence.

5. Resistance to penicillin can be devolped by bacteria, but due to its low toxicity, more than adequate doses of penicillin can be given. It is usually safer from the patient's standpoint to err on the side of overdosage.

6. In infections of the ear, nose and throat, penicillin should be considered when the causative organism is susceptible to this drug.

7. The question of whether penicillin will be given locally or systemically, or both, depends upon circumstances. In acute otitis media it has been given systemically with the aim of avoiding surgery. In osteomyelitis it is given both systemically and locally with the aim of making surgery more successful. In maxillary sinusitis it has been given locally but the prognosis must be cautious, especially in chronic cases.

LILLY RESEARCH LABORATORIES.

REFERENCES

1. New and Nonofficial Remedies, 1944, p. 167.

2. Buxton, Russell, and Kurman, Rachelle: Tetanus; A Report of Two Cases Treated with Penicillin, J. A. M. A. 127:26 (Jan. 6) 1945.

3. Fleming, Alexander: On the Antibacterial Action of Cultures of a Penicillin, With Special Reference to Their Use in the Isolation of B. Influenzae, Brit. J. Exper. Path. 10:226, 1929.

4. Fleming, Alexander: On the Specific Antibacterial Properties of Penicillin and Potassium Tellurite, J. Path. & Bact. 35:831, 1932.

5. Chain, E., Florey, H. W., Gardner, A. D., Heatley, N. G., Jennings, M. A. Orr-Ewing, J., and Sanders, A. G.: Penicillin as a Chemotherapeutic Agent, Lancet 2:226, 1940.

6. Abraham, E. P., Chain, E., Fletcher, C. M., Gardner, A. D., Heatley, N. G., Jennings, M. A., and Florey, H. W.: Further Observations on Penicillin, Lancet 2:177, 1941.

7. Free, A. H., Leonards, J. R., McCullagh, D. R., and Biro, B. E.: The Urinary Excretion of Penicillin After Oral Administration to Normal Human Subjects, Science 100:431 (Nov. 10) 1944.

8. Aagesen, W. J.: Abstract of Discussion on Chemotherapy, J. A. M. A. 126:622 (Nov. 4) 1944.

9. Swanson, C. A., and Baker, D. C., Jr.: The Use of Penicillin in Diseases of the Ear, J. A. M. A. 126:616 (Nov. 4) 1944.

10. Putney, F. J.: Uses of Penicillin in Diseases of the Nose and Throat, J. A. M. A. 126:620 (Nov. 4) 1944.

11. Crowe, S. J., and Ward, A. T.: Local Use of Sulfadiazine, Tyrothricin, Penicillin and Radon in Otolaryngology, North Carolina M. J. 4:431 (Oct.) 1943.

12. Sale, G. G., and Diamond, E. H.: Chronic Suppurative Maxillary Sinusitis Treated Locally with Penicillin by a New Method, Arch. Otolaryng. 40:406 (Nov.) 1944.

13. Delafield, M. E., Straker, Edith, and Topley, W. W. C.: Antiseptic Snuffs, Brit. M. J. 1:145 (Feb. 1) 1941.

14. MacGregor, A. B., and Long, D. A.: The Use of Penicillin Pastilles in Oral Infections, Brit. M. J. 2:686 (Nov. 25) 1944.

XVI

THE FOLLOWING ARE ADAPTATIONS

OF

SEVEN CHARTS

ON

CANCER OF THE LARYNX

EXHIBITED AT THE

Meeting of the American Academy of Ophthalmology and Otolaryngology

by

LE ROY A. SCHALL, M.D.

BOSTON, MASS.

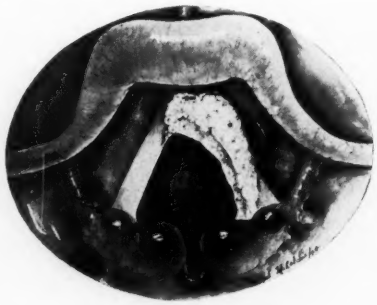
If your patient is *hoarse*

LOOK

It may be CANCER!

Localized *Low Grade* *With Movable Cord*	— *TREATMENT* —	1. *Laryngofissure* (82% *five year cures*) 2. *Radiation*
Localized *High Grade*	— *TREATMENT* —	1. *Radiation*

Extensive *High Grade* *With Movable Cord*	— *TREATMENT* —	*Radiation*



Extensive
Low Grade
With Fixed Cord
Without Adenopathy

— TREATMENT —

Total Laryngectomy

RESULTS—5 year cure 80%

Extrinsic
With Metastases
In Glands

— TREATMENT —

Radiation
(Palliative)

Subglottic With Fixed Cord — *TREATMENT* — *Total Laryngectomy*

CANCER OF THE LARYNX

No Treatment

50% dead in 1 year

95% dead in 2 years

Proper Treatment

80-82% five year cure

MAKE THE DIAGNOSIS EARLY

XVII

THE ALNICO MAGNET—AN AID TO BRONCHOSCOPY AND ESOPHAGOSCOPY

MURDOCK EQUEN, M.D.

ATLANTA, GA.

The dangerous consequences with which foreign bodies of the air and food passages are so fraught demand simpler, more rapid, and easier methods of removal. Asphyxiation from blocking of the air passages, ulceration with perforation of the esophagus are two of the most common immediate dangers; death from diffuse tracheobronchitis from certain substances such as peanut kernels in the lungs is a complication that may occur after a few days, and bronchial stenosis and pulmonary abscesses are likely to develop if the foreign body is retained for some time.

Metallic substances constitute a large part of foreign bodies ingested or aspirated. One of the most frequent of these is an open safety pin which presents several mechanical problems. Attempts at removal of the pin by surgeons even though experienced in bronchoscopy and esophagoscopy frequently traumatize the tissues and occasionally may cause perforations.

The magnet presented herewith fulfills the need for an instrument that will easily remove a magnetizable foreign body and, unlike grasping forceps and other similar bronchoscopic and esophagoscopic instruments, can cause no damage to the tissues. The attraction of the magnet, powerful enough to pull the foreign body along against moderate resistance, will not cause a sharp point to go through the muscle layer of hollow viscera. It is a permanent type magnet, no wires or batteries being used, and is made of alnico.

Alnico is a dispersion hardening alloy of

Aluminum	8%
Nickel	14%
Cobalt	24%
Copper	3%
Iron	51%

Fig. 1.—Magnet for use in the tracheobronchial tree, and magnet for use in the esophagus and stomach, shown actual size.

It has been developed and manufactured by the General Electric Company of Schenectady, New York, and the most recent grade which has been made available is cast alnico V. The quality of a permanent magnet material is usually gauged by three properties of the material—the residual induction, the coercive force, and the maximum external energy value. Cast alnico V has three times the stored magnetic energy of the other alnicos, which themselves possess far greater magnetic energy than any type of permanent magnets known.

The characteristics of alnico V are:

Residual induction	12,500
Coercive force	550
External energy	4,500,000

The magnet for use in the esophagus and the stomach (Fig. 1) is cylindrical, 5.0 cm. in length, 0.5 cm. in diameter, and is attached to a Levin tube, size 12F. To increase the control of the mobility of the tube, a metal stylet has been inserted into the lumen and a small perforation has been made in the tube near the magnet. The other end is attached to a rubber bulb and a diaphragm, so that the stomach can be inflated by pumping air down through the Levin tube.

The magnet used in the tracheobronchial tree (Fig. 1) is shaped somewhat like a match, being cylindrical and measuring 4.0 cm. in length and 0.3 cm. in diameter, and it is attached to a ureteral catheter, and may be passed through the bronchoscope under fluoroscopic guidance.

The procedure of using the magnet can be presented more graphically by giving a typical case report.

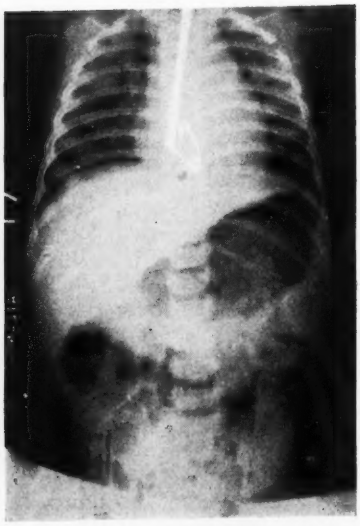


Fig. 2.

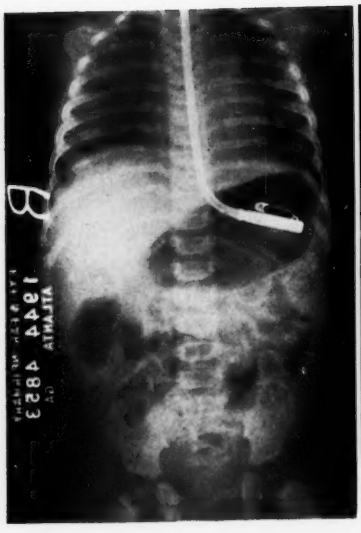


Fig. 3.

Fig. 4.

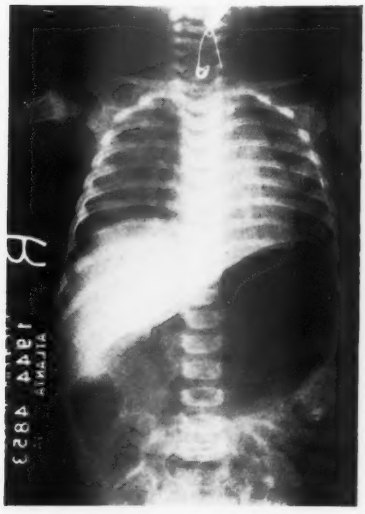


Fig. 5.

REPORT OF A CASE

An eight-month-old male infant swallowed a safety pin 16 hours previous to admission. X-ray film shows an open safety pin with bent keeper branch in the upper third of the esophagus. The pointed branch is turned upward, rendering direct removal not feasible. The pin is carried downward through the esophagus by the magnet (Fig. 2) which is in contact with the point, thus insuring against laceration of the epithelium. The pin is passed into the stomach (Fig. 3), which is inflated with air to allow manipulation. In other cases, in which the pin is in the stomach, this inflation removes folds of the stomach which might prevent the pin from reaching the magnet. The elliptical outline of the inflated stomach is quite obvious in this x-ray film. The pin is not in position for removal, for the point is still upward. The magnet is "flipped" in the stomach by manipulation of the Levin tube, the position of the pin being watched with the fluoroscope. When the spring of the pin is the part nearest the esophagus, the magnet is withdrawn from the stomach bringing the pin with it (Fig. 4). In rapid sequence, the pin is drawn through the cricopharyngeal constriction and into the pharynx (Fig. 5). If the magnet will not pull the pin through the resistance of cricopharyngeal constriction, a bronchoscope can be slipped easily into position and forceps used to remove it.

War-time scarcity of minerals has brought about a great change in the magnetic properties of many common items of daily use. Pre-war pennies with a high copper content show no magnetic properties, but pennies minted in 1944 have iron and other metals in them which allow successful extraction with the magnet. Pre-war safety pins

are highly magnetizable but those manufactured now are of plastic and similar materials and are inert magnetically. Fragments from kitchen utensils, such as forks and spoons, may now be removed easily since there is more iron content and less silver.

PONCE DE LEON EYE AND EAR INFIRMARY.

REFERENCES

1. Silber, Samuel, Kaplan, Carl, and Epstein, Bernard: The Use of a Permanent (Alnico) Magnet In the Peroral Removal of a Metallic Foreign Body (Padlock) From the Stomach, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 53:589 (Sept.) 1944.

2. Jackson, Chevalier: Observations on the Pathology of Foreign Bodies in the Air and Food Passages Based on the Analysis of 628 Cases, Surg. Gynec. and Obst. 28:201-261, 1919.

3. Jackson, Chevalier: A Bullet in the Lung, Bronchoscopic Removal with the Aid of Magnetic Fixation, J. A. M. A. 90:1272-1273, 1928.

4. Equen, Murdock: A New Magnet for Foreign Bodies in the Food and Air Passages, J. A. M. A. 127:87 (Jan.) 1945.

XVIII

HEARING TESTING CABINET

J. KENT LEASURE, M.D.

INDIANAPOLIS, IND.

Our postwar planning should include some thought for improving our detection of the loss of hearing. The cabinet to be described has been in my office for some time and has some useful points which seem worthy of description.

The cabinet should not be called soundproof because that term must be reserved for rooms built solidly of hollow blocks with a soundproof door, well insulated, and with no noisy pipes running through the walls. However, for the average private office sufficient sound is screened out by this type of cabinet so that the lesser hearing losses may be detected. The hearing tests usually conducted in the average office will not be sufficiently accurate to detect losses below 20 to 30 decibels, particularly when a masker is used since that usually lowers the curve about 10 decibels. The use of this cabinet alone with a rubber padded receiver on each ear and no masker allows the detection of hearing at the zero line when it is present.

The receivers are connected by a head band and fit tightly over the ears. The phone over the ear not being tested may be used as a masker when the tone being tested becomes so loud that it is heard by the operator through the room or a shadow curve of the other ear may be suspected.

As a rule the responses which result from testing children under eight years of age (depending upon their alertness) are not so reliable when done with the average equipment. However, with a light placed in front of the patient, connected with another over the audiometer and both operated simultaneously by a push button on a cord, more accurate responses are obtained. These lights work off the city current and are independent of the audiometer with all the wiring and fixtures connected with the lower half of the back of the cabinet. The patient is asked to keep the light in front of him lit as long as he hears the tone being tested. Thus a great deal of unintentional flickering of the light is avoided and more reliable responses result, particularly in children as young as six years of age.

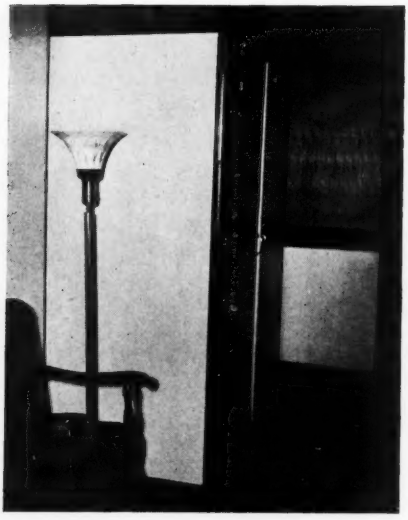


Fig. 1.

The cabinet is too large and heavy to be called portable in the sense of carrying it from place to place as would be required in school work. However, it is constructed in sections so that it may be taken apart and moved when necessary. The height is seven feet, the length six feet and the width is three feet. The walls and top are made of "Weatherwood," a pressed wood pulp half an inch thick. The different sheets are fastened by screws to a wood frame made of strips one inch thick and two inches wide. The sections are held together by screws passed through wood posts at the corners similar to the construction of Newhart's portable room.[1]

The space between the sheets of Weatherwood is divided into squares about three inches in size. The partitions are made of the same material one inch wide placed on edge and joined like egg crate frames. This type of insulation is used by Johns-Manville Company for soundproofing and avoids the settling which might take place when a loose insulating material is used (Fig. 2).

The floor is made of ordinary wood flooring fastened on two by four runners with insulating between. It is not fastened to the cabinet any place but rests on the floor of the room freely.

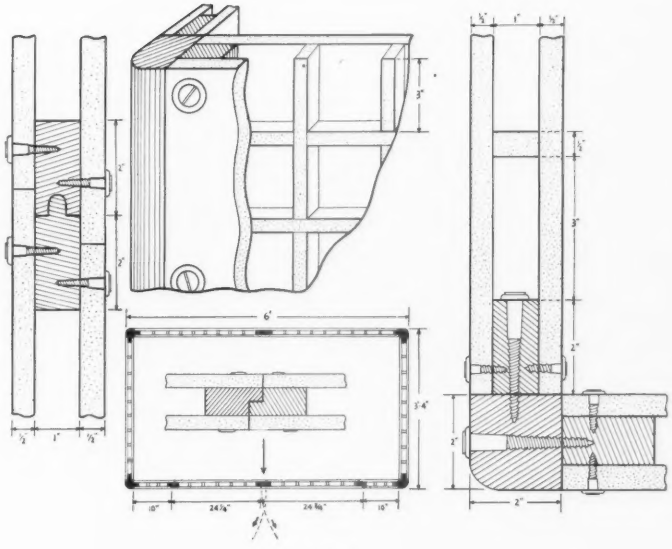


Fig. 2.

Two large doors extend the entire height of one side so that when open they throw the entire side of the cabinet open except a ten-inch strip on either end to which the doors are hinged. If the doors are thrown open between each stage of the test, the air inside may be quickly and completely changed.

The size of the cabinet allows an audiometer on a shelf at one end. The operator stands by it with the patient on a chair facing the other end. There is still enough room in front of the patient and to one side for an observer, particularly a mother.

611 Hume Mansur Building.

REFERENCE

1. Newhart, Horace, and Hartig, H. E.: An Efficient and Practical Booth for Testing Hearing, Arch. Otolaryng. 28:1000 (Dec.) 1938.

XIX

FOREIGN BODIES OF THE MAXILLARY SINUS

JOHN CLAIR HOWARD, JR., M.D.

LIEUT., M.C., U.S.N.R.

KANSAS CITY, MO.

Foreign bodies of the nasal accessory sinuses are comparatively rare but exceedingly interesting to the otolaryngologist, particularly from a surgical standpoint as each case presents a different technical problem that must be worked out by the operator's own ingenuity. A review of the current textbooks on otorhinolaryngology are of meager help as little or no mention is made of this subject. A review of the literature reveals only an occasional case. It is of interest to note that foreign bodies of the nasal accessory sinuses present somewhat of a diagnostic, as well as a surgical problem, as in many of the reported cases the diagnosis of this cause of sinusitis had been overlooked for a considerable period. The importance of diagnosis of this condition is even more forcibly brought home when it is remembered that all too frequently foreign bodies of the sinuses form the basis of malpractice suits, and any prolonged oversight of this condition by the rhinologist may not only damage his professional reputation before a court but may even turn a consultant into a codefendant.

The presence of foreign bodies in the nasal accessory sinuses is mainly due to trauma or follows surgical procedures. Entrance by the route of the natural ostia is exceedingly rare. The foreign body may be almost any conceivable object that will fit into a sinus but by far the most common are roots of teeth, points of operative instruments, such as rasps, and not too infrequently, broken knife blades and bullets may be found. Particular care must be exercised on face wounds in war casualties to make certain slugs have not penetrated the bony walls of the sinus.

Foreign bodies can conceivably find their way into any of the nasal accessory sinuses as has been illustrated in reported cases, but the maxillary sinus is by far the most common location with the ethmoid capsule, the frontal and the sphenoid sinuses following in frequency

The opinions or assertions contained herein are the private ones of the writer and are not to be construed as official or reflecting the views of the Navy Department or the Naval Service at large.

in that order. In traumatic accidents the force necessary to project a foreign body into the frontal and sphenoid sinuses is usually so great that penetration into the brain takes place which leads to a sudden fatal termination, and the problem of such foreign bodies is only of academic interest. Entrance of a foreign body into any of the sinuses, particularly the sphenoid or the frontal, through the natural ostia is remote owing to the anatomic locations of the ostia. However, a foreign body may be introduced accidentally through the natural ostia during surgical procedure.

Sticks and pencils placed in the nasal cavity by children and mentally incompetent adults may penetrate and be broken off in the ethmoid capsule.

The antrum of Highmore is the most common site of lodgement owing, first, to its anatomic location, particularly in relation to the teeth. The formation and growth of the maxillary sinus is in intimate relation to dentition. At birth the antrum measures 8 mm. in length, 4 mm. in height, while in the adult the sinus measures 35 mm. in height and 20 mm. in width. Barnhill[1] states that at birth the upper jaw is shallow and yet all the tooth buds are packed away in it. Packing all these buds in a horizontal line is impossible, so the molar buds are planted in a somewhat perpendicular plane, one bud above the other and all above the posterior end of the alveolus. As the antrum develops, the alveolus is lengthened and the buds are rolled downward and backward and each tooth is erupted in its proper place. If, however, the development of the sinus does not push far enough posteriorly, one or more molar tooth buds will be left in position above the alveolar process where, instead of erupting into the mouth in the normal course of events, one or more molars may be wholly or partly erupted into the antrum of Highmore. Frequently diseased teeth will be separated from the cavity of the maxillary sinus only by the mucous membrane of the sinus because of absorption of the bone covering the root. Thus a molar or a piece of it may be pushed into the antrum during the extraction of the tooth or by outside trauma.

Secondly, foreign bodies in a sinus may result from surgical procedures and the incidence of operative procedure on the maxillary sinus is greater than on all the rest of the sinuses combined. The common office procedures of antrum lavage by one of the standard trocars or punches are a contributing cause.

The maxillary sinus, though protected by stout walls, is in a more exposed position to receive trauma which is the principal cause of foreign bodies.

Any foreign body of the sinuses must be looked upon as a bacteria-contaminated object. This is particularly true in the case of carious tooth roots that may be pushed into the sinus during the extraction of molars.

The severity of the reaction in the sinus depends upon the type of invading bacteria, the condition of the membrane lining the sinus and the anatomic structure of the sinus, particularly in regard to drainage. In general it may be said that the usual reaction to a foreign body takes place. There is an increase in vascularity of the lining membrane due to irritation. Early, there is, because of this stimulus, an increased motility of cilia of the mucous membrane lining the sinus, followed by decreased motility and cessation of movement as inflammation progresses. Attempts to wall off small foreign bodies are occasionally successful and the inflammation subsides. However, in case of failure to wall off the foreign body the acute process continues with suppuration, finally passing from the acute to the chronic stage with a greatly thickened membrane which may progress on to polyp formation. This chronic process lights up from time to time with exacerbations.

The diagnosis of foreign bodies of the nasal sinuses should be based on a careful history, particularly in regard to recent oral surgery, a thorough examination giving particular attention to the oral cavity and an x-ray examination. The x-ray film is not only valuable in revealing the presence of a foreign body and its relation to surrounding structures, but it is indispensable in helping to evaluate the pathology present on which the surgical procedure should be based. The pathology present should govern in a large measure the operative approach to be used. The presence of a foreign body in the sinus should always be considered as an etiological factor in all cases of stubborn resistant sinusitis to which no cause can be assigned. This is particularly true when there is a history of extraction of the upper molar teeth.

REPORT OF CASES

CASE 1. T. C., negro, aged 22, was admitted to the Out-Patient Clinic of the New Orleans Eye, Ear, Nose and Throat Hospital with a complaint of sore throat. He stated that he had been advised to have his tonsils removed. Routine history revealed that the patient had also been having severe headaches following a fight a year previously.

The physical examination revealed the pharynx to be injected, hypertrophic tonsils and a profuse postnasal drip. There was a septal deviation with a large anterior spur. The left nostril was filled with pus and crusts. The nasal cavity was

Fig. 1.—Anteroposterior view, Case 1.

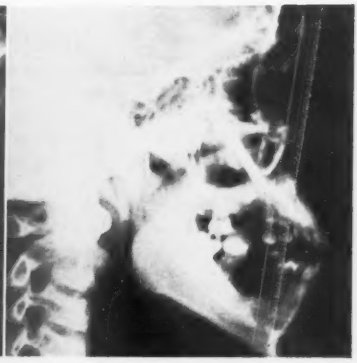


Fig. 2.—Lateral view, Case 1.

cleaned out and a hard metallic object was felt, protruding from the antral wall under the inferior turbinate about one-half inch from its anterior attachment. The metallic object extended across the left nasal cavity and seemed to be imbedded beneath the nasal septum. There was a scar one-half inch long over the zygomatic bone. Further questioning revealed that he had been struck from behind while seated and had been knocked unconscious for 20 minutes. A doctor was called and, after probing the laceration on the cheek, he sutured the wound. There had been severe headaches ever since and a nasal blockage on that side. X-ray examination revealed a knife blade which had penetrated the left zygomatic bone extending through the left maxillary sinus into the left nasal cavity where the point was imbedded in the hard palate beneath the right nasal cavity and antrum (Figs. 1 and 2).

The patient was admitted to the hospital and operated on for the removal of the blade. An incision was made over the point of entrance even though the knife could not be palpated. No evidence of the knife could be found until part of the bone had been chipped away but the blade could not be removed. The maxillary sinus was opened by the Caldwell-Luc technique and the blade was found projecting across the cavity. A large opening was made in the naso-antral wall where the blade penetrated into the nasal cavity. The antrum was filled with pus and the mucous membrane was greatly thickened with polyp formation. A pair of heavy pliers was inserted through the opening in the canine fossa and a firm hold was secured on the shaft of the knife. Further traction was placed on the shank of the blade at its point of entrance and after some little trouble the blade was removed by the author. The polypoid tissue was curetted out and the naso-antral window was enlarged. The knife blade measured 5 3/4 inches and was quite rusty. Tetanus-gas-antitoxin was administered and sulfonamides were given. After a stormy period of 36 hours the patient's recovery was uneventful. He went back to work in three weeks and had been free from headaches when last seen.

Case 2. M. F., aged 56, gave the following history: "While eating a piece of hard candy there was a sudden pain and bleeding and one of my teeth disap-

peared." She was examined by her dentist who probed the tract into her antrum but was unable to recover the tooth. The patient was referred to me 24 hours after the accident. Examination revealed the right upper first molar to be absent and a probe could be passed through its socket into the antrum. This tract was swollen and painful from previous probing. X-ray films showed the presence of the tooth in the antrum. The sinus was otherwise clear. The upper incisors and molars, with the exception of the third molar, were present and while some of them were carious and there was evidence of some pyorrhea, the dentist felt that the teeth could be saved. A large naso-antral opening was made and the tooth removed. The mucous membrane was slightly thickened from past sinus infection but was not diseased enough to warrant removal. The tract from the oral cavity into the maxillary sinus was curretted and silver nitrate applied. Healing was uneventful.

Foreign bodies of the maxillary sinus may be removed by several surgical routes. The procedure used should accomplish the two-fold purpose of removing the foreign body and taking care of the antrum surgically in such a fashion to preserve the future health of the antrum.

Case 1 is one of those freaks that under normal conditions is usually seen but once in a lifetime. However, these bizarre cases may come to our attention more frequently with increase in battle casualties. The surgical procedure of choice in this case included removal of the polypoid tissue from the antrum.

A dental root or a tooth in an antrum may be handled by one of three different methods:

1. Enlarging the tract into the antrum and removal of the foreign body by lavage and suction. A plastic flap is usually made to close the tract. This approach is the one most frequently used by dentists and oral surgeons. Too often this method is used with the sole idea of removal of the foreign body with little consideration of the pathology present in the antrum. As long as infection is present in the antrum, this tract, leading from the oral cavity into the sinus, has a tendency to remain open and serve as an exit for pus; and plastic repair of this tract is usually unsuccessful under these conditions. This procedure is recommended only in the case of a root in a normal antrum, and a normal sinus in the presence of pyorrhea and carious molar teeth is not very common.

2. Removal through a naso-antral opening, which not only produces means for removal of the foreign body but also facilitates the treatment of the infected sinus by increased aeration and drainage. The naso-antral approach allows a view of the lining membrane by direct inspection with a naso-pharyngoscope or antroscope.

3. Removal of foreign body by incision over the canine fossa and removal of part of the anterior wall of the antrum. After removal of the foreign body the remainder of the Caldwell-Luc technique should be carried out with removal of any diseased antral membrane and a large naso-antral opening. This procedure is recommended when the sinus is filled with polypoid tissue but this is heartily condemned as a routine procedure.

In Case 2, the procedure of choice was removal of the foreign body through the naso-antral opening, as a more extensive procedure was not indicated. The Caldwell-Luc procedure is not recommended to be used routinely in cases of this kind because of the danger of injuring the anterior dental nerve which supplies the upper incisors and the possible consequence of dead teeth or at best an unpleasant anesthesia over this area.

Foreign bodies of the nasal accessory sinus with their traumatic etiology may become more common as the war progresses owing to battle casualties and an increase in accidents incident to war work.

SUMMARY

1. Foreign bodies of the nasal accessory sinuses are discussed and two cases of foreign body in the maxillary sinuses are presented.

2. The rationale of various surgical techniques for removal of foreign objects in the maxillary sinus is discussed.

1314 PROFESSIONAL BUILDING.

REFERENCE

1. Barnhill, John F.: Surgical Anatomy of the Head and Neck, Philadelphia, William Wood and Company, 1940.

XX

MICROSCOPY OF THE TEMPORAL BONE WITH POLARIZING FILMS

GEORGE KELEMEN, M.D.

BOSTON, MASS.

The difference in the structural appearance of unstained, living, fresh and fixed tissues examined with bright-field, dark-field, ultraviolet, and polarized light, as compared with stained and incinerated tissues has been studied by Gage, Gage-Day and Starett.[3] In the course of their investigations, they have concluded that each method gives a different picture of the same thing. Since each picture is only a partial representation, a mental combination of these parts is necessary to give a composite which more closely resembles the actual picture than any single representation. They emphasize the fact that even with this composite, our understanding of the real structure of living tissues and organs is still appallingly incomplete.

Examination by polarized light proved to be equivalent to a selective staining by demonstrating double refraction against isotropic elements. Polarizing is superior to a stain in so far as there is no change in the original condition of the material. The appearance of any double refractory element changes with its orientation in space; thus by this method more impressive pictures are rendered and more information gained regarding histological structure than by any other means.[10]

Von Ebner[1] was the first to use polarized light in his studies of bone tissue in 1875. He found that the structure of this tissue is based entirely on the distribution and properties of collagenous fibers. Collagenous fiber is characterized by double refraction in which the optical axis corresponds to the axis of the fiber.[9] When sectioned parallel to its axis, the fiber appears opaque between crossed Nicol prisms; it appears bright when sectioned obliquely. A ring of collagenous fibers shows four completely opaque segments and four alternating segments of maximal brilliance. These phenomena make it pos-

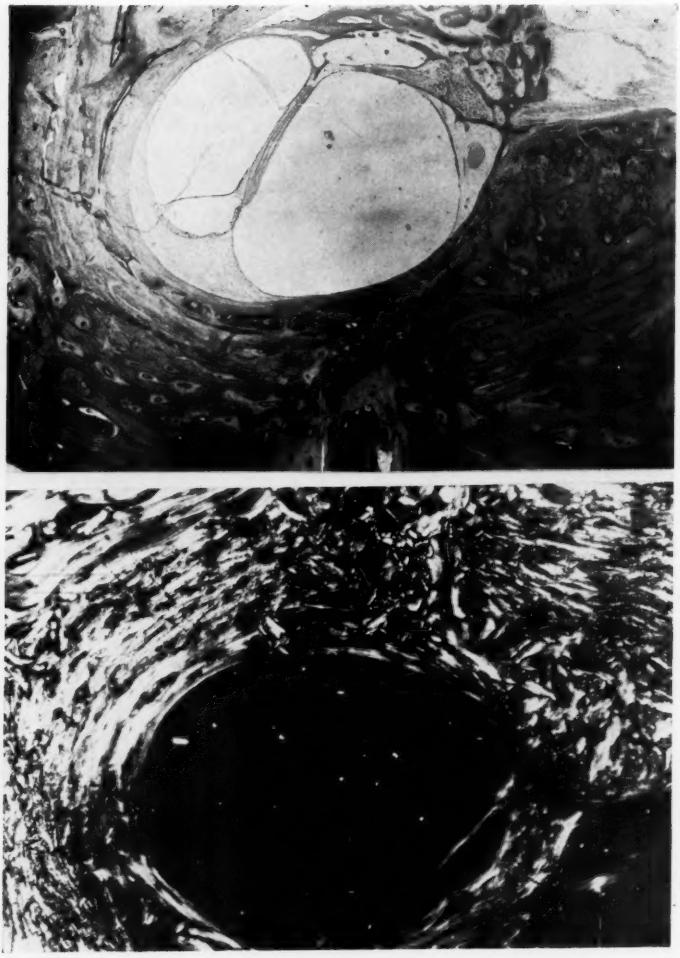
From the Department of Otolaryngology, Tufts College Medical School and the Massachusetts Eye and Ear Infirmary. Aided by a grant from the Charlton Fund, Tufts College Medical School.

sible to follow the path of collagenous fibers. Cross sections of osteons (haversian systems) present the appearance of "maltese crosses".

Two kinds of bone were so identified; a weblike fibrillar bone (called geflechtartiger Knochen by von Ebner[1]) and lamellar bone. In the embryonic period, the entire skeleton of mammals is composed of this weblike fibrillar bone, so-called membranous bone; it resembles a frozen connective tissue.[8] This is replaced in the course of the first years of life by lamellar bone. Destruction of fibrillar bone and replacement by lamellae produce a "breccia." This term, taken from petrology, designates a "rock composed of angular fragments of moderate or large size, cemented together" (Webster). This kind of bone is characteristic of the human skeleton as compared with those of other mammals whose bony architecture is more regular. The whole process is more easily observed by polarized light. By this method is seen the debris which results in the course of destruction and is cemented together in the breccia. The winding of fibers around vessels, later replaced by lamellae and representing a haversian unit, is likewise more clearly demonstrated by polarized light than by any other method; one can also differentiate the collagen in the organs with greater assurance in this way since specific staining methods for collagen will stain besides collagen many other elements.[8]

The information obtained by the use of polarized light in studying the bone structure was first applied to the special conditions prevailing in the bony capsule of the inner ear by Meyer[7] in 1927. He found that fibrillar bone, an immature element which disappears from the entire skeleton around the third year of life, remains preserved in the labyrinthine capsule even to old age. These findings, in addition to including remnants of embryonic cartilage, are characteristic of this area. On the other hand, in contrast to the entire remaining skeleton, no breccia is found in the normal labyrinthine capsule. Complete analysis of these conditions is only possible by observation with polarized light.

The use of polarized light in investigating the problems of otosclerosis was first suggested by Weber[11] in 1930. His analysis was centered on the peculiar formation of the breccia. Although it is not found in the labyrinthine capsule under normal conditions, it is a typical element in otosclerosis; the focus represents in its greatest part a breccia, which however is often not recognized as such on hematoxylin-eosin stained sections. Hematoxylin will demonstrate mainly the cementing lines (Kittlinien) and these lines may be absent at the edge of the focus as well as in its center.



Figs. 1*a* and 1 *b*.—White female, 32 years, cochlear capsule. *a*: hematoxylin-eosin stained, full light; *b*: unstained, polarized light. 24 μ.

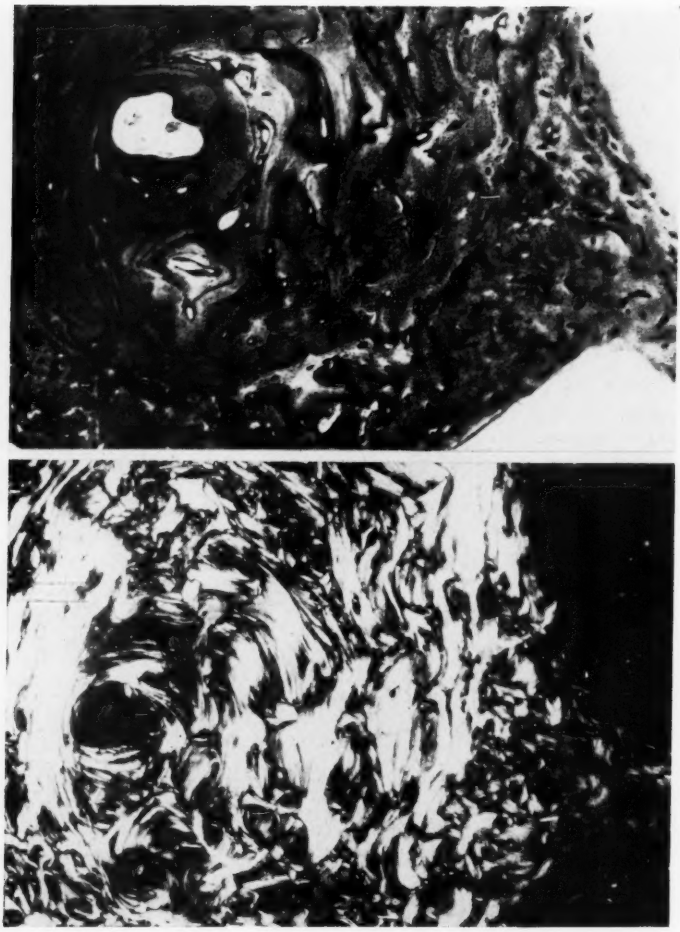
Even with frequent application of other selective histological methods, the study of osseous changes in the inner ear has remained principally a "hematoxylin-eosin-pathology". In this connection, polarized light is infrequently used because of technical difficulties. Preparation of the section to be observed by this method is easy, but to procure and handle the necessary polarizing apparatus may present some difficulty.

Some precaution, however, is necessary even in the preparation of the sections. Fixation in alcohol, formol, picric acid or sublimate is permissible, but chromic acid destroys the double refractory property.[8] This peculiarity of chromic acid must be considered as a contraindication to its use in decalcification and with the osmium methods to demonstrate fat. After decalcification in a five per cent nitric acid solution, paraffin, celloidin, or gelatine may be used for embedding, but after sectioning, those substances, such as paraffin, in which there may be double refraction should be carefully removed. Frozen sections may be used after decalcification. According to Meyer,[7] any section between 5 and 40 μ may be used. Examination is best carried out under water, alcohol, or, preferably, in a ten per cent solution of sodium chloride. When closed with wax, sections in sodium chloride can be preserved for a long time. Binocular observation is preferable. Approaching higher magnifications, contrasts are less well defined, since color components of the polarized light assume an increasing significance.

Polarizing equipment presents considerable difficulties. Usually every type of microscope has its own polarizing attachments; these are always expensive and not everywhere available. The special polarizing microscopes which eliminate several disadvantages inherent to simple polarizing attachments by the insertion of anastigmatic Nicol prisms within the tube of the shaft and other improvements are more expensive and consequently more rare. The small size of the Nicol prisms restricts greatly the visual field.

With the use of light-polarizing materials, many of these difficulties can be overcome. The material used is a film of cellulose acetate with innumerable little needle-shaped crystals embedded in it[4] and bonded between sheets of clear cellulose acetate safety film. It can be manufactured in sheets of any size. The small amount of film necessary entails a negligible expense. We have used it in the following manner:

Microscopy. A piece of film was cut corresponding to the size of the slide and placed under the slide carrying the unstained section;



Figs. 2*a* and 2*b*.—Same series as Fig. 1*a* and 1*b* Labyrinthine nucleus. *a*: hematoxylin-eosin stained, full light; *b*: unstained, polarized light. 24 μ.

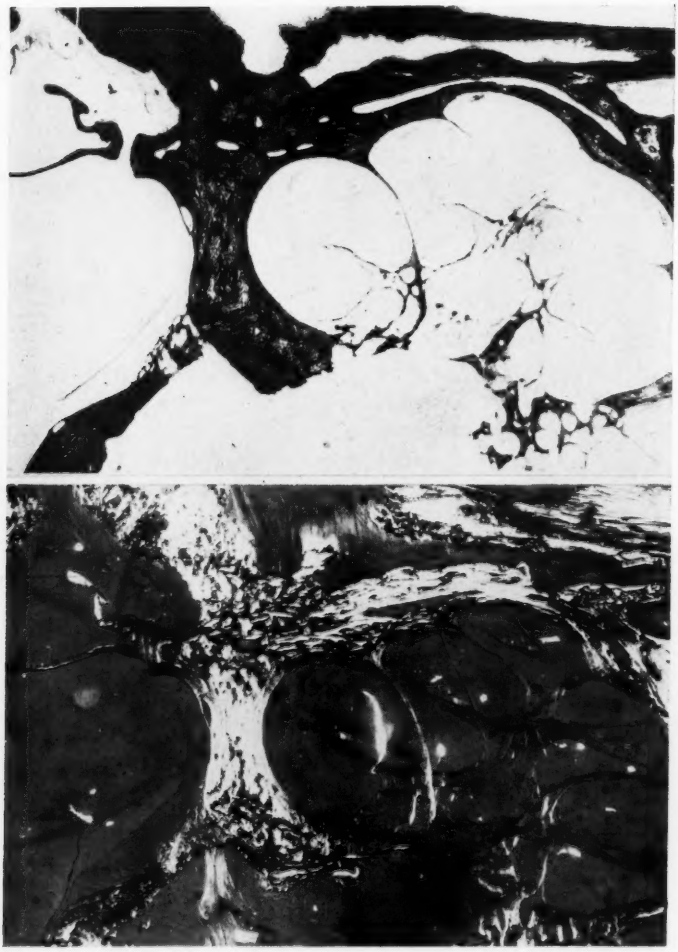
these were clamped together to the stage of the microscope. This piece of film served as a polarizer. A second small round piece was then cut and dropped onto the inner diaphragm of the ocular after it had been temporarily unscrewed. This piece served as an analyzer. Simple rotation of the ocular in the shaft produced opaque and bright positions. For the sake of easier orientation in the unstained section, it is advisable to reduce the iris diaphragm of the substage-condenser almost to a pinpoint aperture, while when turning into the polarizing position a somewhat larger opening may be found useful. The visual field remains entirely unrestricted by this method of polarization and is merely limited by the optical system of the microscope itself. In this way rapid survey of the entire section is greatly facilitated.

Photomicrography. One round piece of the film was fastened in front of the objective and remained immobile. A larger piece was slipped into a filter carrier frame between the microscope and the source of light. This piece could be easily rotated. Since with the use of Nicol prisms or any other polarizing material, there is a loss of more than 50 per cent of illumination, the time of exposure must be longer as, for example, in the case of a section of the same thickness but stained with hematoxylin-eosin and photographed in full light.

To eliminate as many media with different refractory properties as possible, slides or coverslides made of polarizing material would be desirable. By inserting a round piece of polarizing sheet into the frame under the condenser which will carry colored filters, the polarizing process would be reduced to a minimal maneuver. This is of importance when examination of a long series of sections is involved.

When one is working on histological problems concerning the temporal bone, a wide field stereoscopic microscope for rapid orientation in the long series and for stereophotography[5] of sections is indispensable, besides the microscope used for the routine examination and photomicrography. It is welcome knowledge that a third, the polarizing, microscope can be replaced by a device of greatest simplicity without additional expense.

To demonstrate the results obtained by using polarizing sheets, six photomicrographs are presented. The three pairs illustrate the contrast between sections stained with hematoxylin-eosin photographed in unpolarized light, and unstained sections photographed in polarized light. In every instance the temporal bone was sectioned in a horizontal plane. The unstained and the hematoxylin-eosin stained sections represent adjacent links of the series; thus, their details are comparable.



Figs. 3*a* and 3*b*.—Macacus rhesus, cochlear capsule, *a*: hematoxylin-eosin stained, full light; *b*: unstained, polarized light. 24 μ.

In case the reproduction of strictly identical elements is desired, the unstained section must be photographed first in polarized light and then again in full light after staining. When a stained section contains details which should be observed by polarizing, the section must be decolorized. For the purpose of this study, reproduction of two adjacent sections seemed to be sufficient.

The three pairs of pictures show the temporal bone in an adult, and in a monkey (Macacus rhesus). For the adult, one pair represents the cochlear capsule and one the nucleus of the labyrinth, the solid block between the semicircular canals. The same magnification (about 25 times with a Zeiss Planar) was used in all the pictures. The slides had to be exposed to the polarized light at least five times longer than the hematoxylin-eosin stained sections, using the full light of the same lamp.

Figs. 1A and 1B demonstrate the special skein-like bone tissue (Straehnenknochen of Meyer[7]) with its mat of manifold interwoven fibers in a white female, aged 32, who died of meningitis. Not only do the main tracts of this mat differ in their size, but much variety is seen in its components as the bundles are made up from fibers of varying thicknesses.

Figs. 2A and 2B taken from the labyrinthine nucleus of the same series clearly show the internal structure of the haversian systems. Coarser and finer fibrils, sometimes in broad braids, can be followed as they wind around the blood vessels. Their oblique course can be seen to best advantage where the fiber in its course changes several times from opacity to brightness.

Figs. 3A and 3B demonstrate the cochlear capsule of a Macacus monkey (12-15 months old). In this animal the compacta of the bone in the internal ear is much reduced as compared to that of humans;[6] there is a much higher degree of pneumatization. The bony walls between the cavities of the osseous labyrinth and the septa between the pneumatic spaces are very delicate. Haag emphasized that remnants of embryonic elements, endoglobular spaces, and skein-like bone are most commonly seen in the region around the windows. This is substantiated by Fig. 3B where rich fibrillar differentiation is seen in the wall of the cochlear capsule around the boundary between the basal and middle cochlear turns adjacent to the stapes plate. The stereoscopic appearance of the basilar organ produced by the polarized light is remarkable.

SUMMARY

Polarized light is a great aid in analyzing structural details in general bone histology under normal and pathological conditions.

This method reveals certain conditions in the osseous inner ear capsule which are characteristic of this region and seen in no other area of the human skeleton.

Polarizing apparatus is expensive and not always available. In the greater part of the examinations (except those where the requirements are of a very special nature), it can be replaced by polarizing films which offer a method of extreme simplicity at negligible expense. One particular advantage as against the Nicol prisms is the unlimited visual field. This is especially valuable in photomicrography.

In the routine histological analysis of the temporal bone, it is desirable to include observation by polarized light. This is facilitated by the use of polarizing films.

The six sections of temporal bone pictured here belong to the collection of Dorothy Wolff, Ph.D., whose kindness in permitting their use is gratefully acknowledged.

Richard W. St. Clair, head of the photographic laboratory of the Massachusetts General Hospital, rendered valuable technical assistance in photomicrography.

414 Beacon Street.

REFERENCES

1. von Ebner, Victor: Untersuchungen ueber das Verhalten des Knochengewebes im polarisierten Licht, Sitzungsber. Kais. Akad. Wiss. Wien, 3. Abt. 70:105-143, 1874.

2. Gage, S. H.: The Microscope, 17th ed., Ithaca, N. Y., The Compton Publishing Co., 1943, p. 172.

3. Gage, S. H., Gage-Day, Mary, and Starett, C. C.: The Structural Appearance of Unstained, Living, Fresh and Fixed Tissues and Organs Revealed by the Bright Field, the Dark Field, the Ultra-violet, and the Polarizing Microscope in Comparison with Stained and with Incinerated Tissues and Organs, Anat. Rec. 55:4, Suppl. 17, 1933.

4. Grabau, Martin: Introduction to Polarized Light and Its Application, Cambridge, Mass., Polaroid Corporation, 1940.

5. Gray, A. A.: Methods of Preparing Stereoscopic Photomicrographs of the Inner Ear, Acta Oto-Laryng. 15:408-413, 1931.

6. Haag, Erhard: Ueber den Bau der Labyrinthkapsel einiger Affenarten, Arch. f. Ohren-, Nasen-u. Kehlkopfkr. 136:14-23, 1933.

7. Meyer, Max: Ueber eine eigentuemliche Art von Knochengewebe beim erwachsenen Menschen (den lamellenlosen, feinfaserigen (straehnenartigen) Markknochen) und ueber den embryonalen Markknochen, Ztschr. f. d. ges. Anat. Abt. 1, 83:734-751 (Aug.) 1927.

8. Petersen, Hans: Histologie und mikroskopische Anatomie, Münschen, J. F. Bergmann, 1924, Vol. 3, pp. 214-262.

9. Petersen, Hans: Die Organe des Skelettsystems. In: von Moellendorff, Wilhelm; Handb. d. mikrosk. Anat. d. Menschen, Berlin, J. Springer, II. 2:521-678, 1930.

10. Schmidt, W. J.: Polarisationsmikroskopie. In: Péterfi, Tibor: Methodik d. wiss. Biologie, Berlin, J. Springer, 1928, I. Allgemeine Morphologie, pp. 380-417.

11. Weber, Moritz: Otosklerose im polarisierten Lichte. (Zur Frage der Fibrillen und Kittlinien.) Ztschr. f. Hals-, Nasen-u. Ohrenh. 23:415-450 (July) 1931.

Clinical Notes

XXI

MASSIVE PHARYNGEAL HEMORRHAGES

LIGATION OF THE EXTERNAL AND COMMON CAROTID ARTERIES

REPORT OF A CASE

GEORGE H. WOODRUFF, M.D.

JOLIET, ILL.

Doubtless the serious complications of peritonsillar and parapharyngeal abscesses have become less common with the widespread use of the sulfonamides, but they will continue to be seen from time to time, especially in neglected cases.

Following is an account of a case of serious pharyngeal infection in which three massive hemorrhages occurred. Because I did not see the patient until the suppurative process had run its course, I hesitate to name the location of the suppuration; but my impression is that it was in the peritonsillar and parapharyngeal spaces.

On March 11, 1943, a boy of 17 years developed a sore throat on the right side which rapidly became very severe, so that in a few hours there was some swelling of the neck in addition to considerable swelling in the throat. He stayed in bed for the next three days, but on the 15th, five days after the onset, he went back to work driving a truck. It was damp and rainy and his neck became stiff and the right ear began to ache. On the 17th he became very sick and could not get home. He spent the night in a gas station, came home early in the morning of the 18th and went to bed. There was fever, pain, considerable swelling, and marked tenderness of the neck below the right ear. They thought it might be mumps and called Dr. A. who saw him at 5:00 a. m. on the 19th, left him some pills and said he had a very sore throat.

Read at a meeting of the Chicago Laryngological and Otological Society, December 4, 1944.

The doctor returned in the afternoon and told them to use an electric pad. He visited the patient daily up to and including the 26th. By then the neck swelling was very large and the pain terribly severe. On the 27th Dr. A. could not come to see him so they called Dr. B. who came at 11:00 a.m. and wanted to lance the abscess, either in the throat or externally, but the patient refused. Dr. A. came at 1:30 p. m. and while he was using a wooden tongue depressor the abscess ruptured, after which the patient spit up a considerable amount of pus and felt better. At 6:30 p. m. the same day there was a sudden hemorrhage amounting to approximately two quarts according to the mother's estimate. They could not get Dr. A. so they called Dr. B. and also a nurse. The patient was taken to the hospital at Morris at 8:30 p. m. and on arrival there was very pale and blue. He was given vitamin K and put to bed in the hall because there were no vacant rooms. The temperature on admission was 99° F. and the next day it varied from 99° to 102.4.° During the next three days it came down by lysis until on April 1 it was about one degree above normal. During this time his medication consisted of sulfathiazole gm. 1 four times a day, aspirin gr. 10 three times a day and an occasional dose of amytal for restlessness.

On April 1 the patient felt quite well but about 11:00 a. m. there was a sudden gush of blood which the mother stated filled three large emesis basins. It was described on the nursing chart as an emesis of one quart of blood. He was given morphine gr. ¼ and 2 cc. of thromboplastin, and an ice collar was applied. The following day the temperature fell to 96.2° F. at 8:00 a. m. but gradually climbed to 97.4.° His chest was x-rayed and found normal and the blood Kahn test was reported negative.

On April 3 at 3:30 a. m. there was another large emesis of blood. Vitamin K and thromboplastin were again given. At 6:00 a. m. the temperature was 96° F. and at 10:00 a. m. 500 cc. of citrated blood was given, and 350 cc. of physiologic sodium chloride solution was added later.

On the 4th the temperature rose to 102°, probably due to a reaction from the transfusion, but rapidly fell to normal. The patient felt well most of the day but toward evening became very nervous and manifested great fear of another hemorrhage. His pupils were dilated and the pulse rapid and pounding and he continued to be restless all evening until morphine gr. ⅛ was given at 9:00 p. m.

I saw the patient for the first time at 8:30 a. m. the next day, April 5, in the Morris Hospital. He was a tall, slender youth who,

with the exception of a marked pallor, did not appear acutely ill. At this time there was only slight swelling and tenderness of the right neck. Pharyngeal swelling was also very moderate and confined to the right peritonsillar area. There was little or no soreness on swallowing, but the boy had a feeling that another hemorrhage was threatening and this kept him very apprehensive. His blood count was: red blood count, 2,250,000; hemoglobin, 42%; and white blood count, 17,000.

Because of the history of three massive hemorrhages within one week and because it was felt that there was no assurance that another might not occur and prove fatal, operation was advised and consented to. This was carried out immediately.

The incision was made along the anterior border of the sternomastoid muscle and the carotid sheath was exposed in the main by blunt dissection.

The dissection was not easy because the tissues appeared to be matted together; the large vessels were exposed and isolated with some difficulty and the external carotid was then ligated. The blunt dissection was continued upward until the posterior compartment of the parapharyngeal space was exposed. No free pus was found, but considerable old clotted blood was removed. This area was gently explored with the index finger and a copious rapid bleeding started up which appeared to originate at about the level of the tonsil, but I was not able to identify the bleeding vessel. The hemorrhage was stopped by digital pressure in the upper portion of the wound and while this pressure was maintained the common carotid artery was ligated with two silk ligatures. Upon tying the ligatures the digital pressure was released and the bleeding did not recur. A rubber tissue drain was inserted, the muscle and fascial layers closed with catgut and the skin wound was closed with black silk.

The patient was returned to bed and 500 cc. of citrated blood was given by Dr. B.

From this time his convalescence, with the exception of moderate postoperative emesis of bloody mucus, was steady and uneventful and he left the hospital on April 15, 36 days after the onset of the illness and 10 days after the operation. The blood count the day before dismissal was: red blood count, 3,580,000; hemoglobin, 56%; white blood count, 9,750.

I realize that a good many cases of this type have been reported; but, nevertheless, they are infrequent, and a good many men will

probably go through their entire practice without ever encountering this complication. In this case there was apparently considerable unwillingness to believe that the hemorrhage came from the peripharyngeal suppuration and the chest was x-rayed in the search for the site of hemorrhage. It would appear that many practitioners do not realize the possibility of serious hemorrhage resulting from abscesses in the pharyngeal region.

There may be some question whether ligation of the common carotid artery with its inherent morbidity and mortality was justified. However I believe the train of circumstances before and during operation called for it.

This whole subject was thoroughly reviewed and analysed by Salinger and Pearlman[1] in 1933. In cases like this they recommend:

"Exposure of the carotid sheath and if the patient's condition is not too critical a search for the source of the hemorrhage. In a minority of cases one of the branches or the stem of the external carotid artery will be at fault. In this event the external carotid artery or such a branch should be ligated. If one fails to locate such a pathologic change, the only safe procedure is ligation of the common carotid artery."

Their analysis of 227 cases shows that in the majority the lesion was in the internal carotid artery.

Kerwin[2] quotes an article by Greenfield in which he reports the results of an investigation of 33 cases of hemorrhage due to peritonsillar abscess. Postmortem examination in this group showed that most of the hemorrhages came from branches of the external carotid artery and not from the internal carotid artery. However, when the analysis included suppuration in all the connective tissue spaces about the pharynx, as did the review of Salinger and Pearlman, the lesion in the great majority was in the internal carotid artery.

Grove,[3] in 1942, reported a case in which an erosion was found in the ascending pharyngeal artery. This vessel was ligated and the patient recovered.

Salinger and Pearlman[1] as well as others mention an alternative procedure that may be followed in some cases. This is to ligate the external carotid artery and to place a ligature about the common carotid artery leaving the wound wide open. A trained attendant would have to be at the bedside constantly so the ligature could be tied at once in case the hemorrhage recurred. This requirement could not have been met in my case.

It seems to me that one factor in the occurrence of the expulsive hemorrhage at varying periods following incision and evacuation of pus may be the following:

The internal carotid artery or other large vessel may be already seriously eroded but the not inconsiderable pressure of the confined pus and the inflammatory zone surrounding it aids in preventing a complete rupture. Upon the evacuation of pus by incision or spontaneous rupture of the pharyngeal tissues, the weakened vessel wall gives way, its external support being removed.

When an undrained abscess or a phlegmon of the parapharyngeal space causes a break in the wall of an artery a false aneurysm, that is, a collection of blood in the tissues, results. This may be a mixture of blood and pus in the case of a frank abscess. If there is very little pus formation which may happen in the case of a phlegmon, as was pointed out by Mosher[4] and by Brunner,[5] the collection may be nearly pure blood. The size of the false aneurysm is limited by the rising pressure on the outside of the affected vessel. In case the abscess ruptures spontaneously or is incised, the extra-arterial pressure falls and free hemorrhage results. Therefore, if there is any reason to suspect that a large vessel has either ruptured or been seriously weakened, incision through the pharyngeal wall is a very dangerous procedure. This is the reason that Grove[3] suggested the advisability of opening parapharyngeal abscesses by the external route rather than through the pharynx. He makes the point that such a hemorrhage can be temporarily controlled by packing through an external incision. This would be impossible or extremely difficult through a pharyngeal incision.

Perhaps it should be mentioned that in a few cases hemorrhage has recurred even after the ligation of the common carotid artery. Richards[6] reported such a case in 1941. The internal and external carotid arteries were then ligated and the patient recovered. The explanation of the hemorrhage after the common carotid artery was tied was that due to the collateral circulation blood flowed down the external carotid to the bifurcation and then upward into the internal carotid artery.

In conclusion, it appears that the procedure of choice in the great majority of massive pharyngeal hemorrhages is still ligation of the common carotid artery. Rarely it may be necessary to ligate subsequently either the internal or the external carotid artery.

250 North Ottawa Street.

REFERENCES

1. Salinger, S., and Pearlman, S. J.: Hemorrhage from Pharyngeal and Peritonsillar Abscesses, Arch. Otolaryng. 18:464 (Oct.) 1933.

2. Kerwin, R. W.: Carotid Artery Surgery, Arch. Otolaryng. 35:30 (Jan.) 1942.

3. Grove, W. E.: Abscess of the Parapharyngeal Space: Massive Hemorrhage, Recovery, A Case Report, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 51:1053 (Dec.) 1942.

4. Mosher, H. P.: Deep Cervical Abscess and Thrombosis of the Internal Jugular Vein, Laryngoscope 30:365, 1920.

5. Brunner, Hans: Infection of the Parapharyngeal Space, Arch. Otolaryng. 31:597 (April) 1940.

6. Richards, L. G.: Massive Hemorrhage from the Carotid Artery, ANNALS OF OTOLOGY RHINOLOGY, AND LARYNGOLOGY 50:1221 (Dec.) 1941.

Society Proceedings

SIXTY-SIXTH ANNUAL CONGRESS

OF THE

AMERICAN LARYNGOLOGICAL ASSOCIATION

NEW YORK, N. Y., JUNE 7-8, 1944

THE PRESIDENT, DR. CHARLES J. IMPERATORI, PRESIDING

(Abstract of Procedings)

(CONTINUED FROM THE DECEMBER 1944 NUMBER)

Stereoscopic Radiographs vs. Single Films

F. M. LAW, M.D.

NEW YORK, N. Y.

(This paper appeared in full in the September issue, page 531.)

Laryngocele

H. MARSHALL TAYLOR, M. D.

JACKSONVILLE, FLA.

(This paper appeared in full in the September issue, page 536.)

Dr. Chevalier L. Jackson stated that he had never seen a case of laryngocele requiring external operation until recently. The laryngocele was in a young service man and could be demonstrated externally in the neck but was easily reduced. Dr. Jackson expected to operate on this patient in the near future and hoped to present as successful results as obtained by Dr. Taylor. He found planography very successful in demonstrating the extent and location of the sac in this case.

Dr. LeRoy A. Schall reported having seen four cases of laryngocele at the Massachusetts Eye & Ear Infirmary during the past few years. In one case the laryngocele was bilateral. He mentioned hav-

DISCUSSION

ing reported one case recently which was associated with cancer of the larynx, in which a laryngectomy was performed. He had thus far failed to find a similar case in the literature except one reported by Dr. New in an article on cysts of the larynx.

Dr. Schall also mentioned a patient who had an external swelling over the larynx for years, but refused operation, until she finally came to the hospital in a comatose condition. The autopsy specimen showed a combined internal and external laryngocele.

Dr. Samuel Iglauer recalled a case of his, referred to by the essayist. He first saw the child when he was about 8 years of age. Operation was not considered necessary at the time. He saw the patient 20 years later with apparently no increase in the symptoms. The patient entered the army, where he died of disease or injury. Apparently, in the speaker's opinion, unless a laryngocele produces symptoms, there is no particular need for operation.

Dr. Iglauer felt that the term "laryngocele" was unfortunate, since the protrusion is really an aerocele. Commenting on Dr. Taylor's statement that these sacs are a sort of accessory lung, such as found in apes, and that when the animal closes his glottis he has a quantity of air trapped on which he can draw for oxygen, in the speaker's opinion there could be very little absorption of oxygen from a sac of that size. He suggested that in the animal this sac might possibly serve as a life preserver when in the water or, as he had read somewhere, could be blown up to scare possible enemies. He was unwilling, however, to express an opinion on the matter.

Concerning the statistics cited, Dr. Iglauer believed that the 85 cases collected by Moore (referred to by the essayist) was inaccurate, because Moore was dealing with various other larynx lesions among which there were only 10 or 12 cases of pure laryngocele.

As to diagnosis, Dr. Iglauer was satisfied with x-ray films taken while the patient blows up the sac by doing the Valsalva act. The same effect can be obtained in a child by giving him a whistle to blow. He saw no objection to putting a needle into the sac for diagnosis, although if the sac did become infected one would have a serious neck infection to deal with.

Dr. W. L. Simpson recalled his case, which Dr. Taylor mentioned in the paper, and which was of traumatic origin, the patient having

been struck in the neck. At operation an opening was found in the left thyroid cartilage, the size of a small egg, through which the mass could be pressed back into the larynx and then reblown by the patient. He also mentioned the use of lipiodol for more accurate delineation of the sac.

Dr. George B. Wood stated that, although he had personally never seen a case of laryngocele, he had always taught that there were two types, namely, the traumatic and the idiopathic. They are differentiated by the fact that in the former the air is in the connective tissue, with an inflammatory capsule about it, whereas in the idiopathic type the sac is lined with epithelium.

Dr. Charles J. Imperatori mentioned having seen a case of this kind about 30 years ago, when their x-ray appartus was either absent or nonfunctioning, and in which the diagnosis was arrived at by palpation and auscultation. He felt that the maneuver whereby the larynx is moved from side to side, producing normally a crepitus, is one that has definite value and should be employed more frequently.

Dr. F. M. Law thought planographs or plain lateral films are satisfactory means of revealing the mass which, being filled with air, is easily outlined in the dense tissues of the neck. Pictures should be taken on both inspiration and expiration.

Dr. Taylor, in closing, referred Dr. Iglauer to Negus' book on the mechanism of the larynx for an answer to his suggestions in physiology. As for the sac acting as a lifesaver in the ape, the theory was untenable because that animal is far from being aquatic, making no effort to swim when put into the water and drowning without effectual effort at saving himself.

Pathology of Nasal Mucous Membrane and Suggestions as to Treatment

LEROY A. SCHALL, M.D.

BOSTON, MASS.

(This paper appeared in full in the September issue, page 391.)

DISCUSSION

Dr. Arthur W. Proetz was in agreement with Dr. Schall and heartily seconded his suggestions as to treatment. Too much emphasis cannot be placed on the simple procedures which have stood the test

DISCUSSION

ing reported one case recently which was associated with cancer of the larynx, in which a laryngectomy was performed. He had thus far failed to find a similar case in the literature except one reported by Dr. New in an article on cysts of the larynx.

Dr. Schall also mentioned a patient who had an external swelling over the larynx for years, but refused operation, until she finally came to the hospital in a comatose condition. The autopsy specimen showed a combined internal and external laryngocele.

Dr. Samuel Iglauer recalled a case of his, referred to by the essayist. He first saw the child when he was about 8 years of age. Operation was not considered necessary at the time. He saw the patient 20 years later with apparently no increase in the symptoms. The patient entered the army, where he died of disease or injury. Apparently, in the speaker's opinion, unless a laryngocele produces symptoms, there is no particular need for operation.

Dr. Iglauer felt that the term "laryngocele" was unfortunate, since the protrusion is really an aerocele. Commenting on Dr. Taylor's statement that these sacs are a sort of accessory lung, such as found in apes, and that when the animal closes his glottis he has a quantity of air trapped on which he can draw for oxygen, in the speaker's opinion there could be very little absorption of oxygen from a sac of that size. He suggested that in the animal this sac might possibly serve as a life preserver when in the water or, as he had read somewhere, could be blown up to scare possible enemies. He was unwilling, however, to express an opinion on the matter.

Concerning the statistics cited, Dr. Iglauer believed that the 85 cases collected by Moore (referred to by the essayist) was inaccurate, because Moore was dealing with various other larynx lesions among which there were only 10 or 12 cases of pure laryngocele.

As to diagnosis, Dr. Iglauer was satisfied with x-ray films taken while the patient blows up the sac by doing the Valsalva act. The same effect can be obtained in a child by giving him a whistle to blow. He saw no objection to putting a needle into the sac for diagnosis, although if the sac did become infected one would have a serious neck infection to deal with.

Dr. W. L. Simpson recalled his case, which Dr. Taylor mentioned in the paper, and which was of traumatic origin, the patient having

been struck in the neck. At operation an opening was found in the left thyroid cartilage, the size of a small egg, through which the mass could be pressed back into the larynx and then reblown by the patient. He also mentioned the use of lipiodol for more accurate delineation of the sac.

Dr. George B. Wood stated that, although he had personally never seen a case of laryngocele, he had always taught that there were two types, namely, the traumatic and the idiopathic. They are differentiated by the fact that in the former the air is in the connective tissue, with an inflammatory capsule about it, whereas in the idiopathic type the sac is lined with epithelium.

Dr. Charles J. Imperatori mentioned having seen a case of this kind about 30 years ago, when their x-ray appartus was either absent or nonfunctioning, and in which the diagnosis was arrived at by palpation and auscultation. He felt that the maneuver whereby the larynx is moved from side to side, producing normally a crepitus, is one that has definite value and should be employed more frequently.

Dr. F. M. Law thought planographs or plain lateral films are satisfactory means of revealing the mass which, being filled with air, is easily outlined in the dense tissues of the neck. Pictures should be taken on both inspiration and expiration.

Dr. Taylor, in closing, referred Dr. Iglauer to Negus' book on the mechanism of the larynx for an answer to his suggestions in physiology. As for the sac acting as a lifesaver in the ape, the theory was untenable because that animal is far from being aquatic, making no effort to swim when put into the water and drowning without effectual effort at saving himself.

Pathology of Nasal Mucous Membrane and Suggestions as to Treatment

LEROY A. SCHALL, M.D.

BOSTON, MASS.

(This paper appeared in full in the September issue, page 391.)

DISCUSSION

Dr. Arthur W. Proetz was in agreement with Dr. Schall and heartily seconded his suggestions as to treatment. Too much emphasis cannot be placed on the simple procedures which have stood the test

of time and proven their reliability. It is always well that someone sound the note of warning against being misled by fancy therapeutic measures backed by high pressure propaganda.

Dr. Proetz was not in favor of nasal irrigation, but prefers capillary suction with a slender tube which enables one to draw secretions from the meatuses that cannot be reached by irrigation. He is opposed to mass suction with nostrils occluded. He offered a simple procedure for the relief of the dry nose, namely, a 5 per cent glycerine, 5 per cent alcohol solution in normal saline. A few drops of spirits eau de cologne are added and the solution is stained with sunset yellow. It is stimulating, deodorant and very effective when used three or four times daily, half a dropper full in each nostril. The alcohol induces a local hyperemia and stimulates the flow of mucus. Dr. Proetz also has found it useful in the bronchi of children with tracheobronchitis associated with thick secretion.

Dr. John H. Foster called attention to the use of ethylene disulfonate as reported in the *Journal of Pediatrics*. Its use is based on the stimulation of carbohydrate metabolism. The speaker and Dr. Cody had experimented with the use of dinitrous phenol with excellent results in a number of cases, but had given it up, fearing the drug might prove dangerous. His experience with operative narrowing of the nasal chambers in atrophic rhinitis had been favorable, and he was surprised that Dr. Schall had had no luck with the operation. Dr. Foster also mentioned good results from the prolonged use of cod liver oil in atrophic cases.

Dr. Charles J. Imperatori reported his experience in a family of four girls, ages 7 to 14, and the parents, all suffering from atrophic rhinitis. Using estrogenic substance following irrigation, the degree of improvement was in inverse ratio to the age; that is, the greatest relief was in the youngest and the least in the oldest.

Dr. Thomas E. Carmody thought there might be some value in the modern methods of dress, which permits greater exposure of the skin. He agreed with Dr. Schall on the virtue of rest in bed and Dover's powder in acute conditions. He was not convinced, however, that the habit of sleeping in a cold room with windows wide open was beneficial. He favored the limited use of silver salts, but warned against prolonged use, having seen cases of argyria result. He thought the neosilvol solution dangerous because its pale color is deceiving. Dr. Carmody agreed with Dr. Proetz in the usefulness of capillary suction and is also opposed to routine irrigation.

Dr. Thomas C. Galloway believed that we should not be too hasty to condemn certain procedures which in some hands have proven beneficial. By studying the processes of inflammation and nature's method of overcoming them, we frequently find certain measures useful which assist and hasten them. Just as x-ray therapy heightens the intensity of an acute inflammation and hastens resolution, so the judicious use of packs, hypertonic magnesium sulfate, and partially ionized silver salts may accomplish the same thing in the nose.

Dr. Frederick Z. Havens mentioned a report of investigations in heating and ventilation which proved that in the average constructed building there was sufficient infiltration of air to provide a complete interchange between inside and outside seven times an hour. This fact should put the quietus on the fresh air open-window fetish.

Dr. Schall, in closing, agreed as to the value of capillary or spot suction. Yet at certain times irrigation and packs may be very useful provided the therapy is not overdone.

Replying to Dr. Foster, he had had no experience with the ethylene disulfonate.

Inter-American Relations in Otolaryngology

C. L. Jackson, M.D.

Philadelphia, Pa.

(This paper appeared in full in the September issue, page 544.)

DISCUSSION

Dr. Ralph Fenton complimented Dr. Jackson on the industry and care with which he and his Committee had worked on the plans for the proposed Congress. He felt that we in the United States had much to gain from the experience of our Latin American colleagues, and that a free interchange of ideas between us should prove a great stimulus for all of us.

Dr. Imperatori agreed heartily with Dr. Fenton, and commented on the utter lack of understanding that exists in many circles. Differences of temperament will prove no obstacle once personal contact is established. In fact, these differences will, in the long run, be found mostly imaginary and fictional. Our aims are identical and nothing but good can result from closer collaboration.

Some Problems in the Surgical Treatment of Bilateral Abductor Paralysis of the Larynx

JOSEPH D. KELLY, M.D.

NEW YORK, N. Y.

(This paper appeared in full in the September issue, page 461.)

DISCUSSION

Dr. John H. Foster inquired whether it would be of any advantage in the healing for the patient to wear a rubber mold tube such as the Jacksons recommended for dilatation of strictures.

Dr. J. W. McCall stressed the importance of identifying and fixing the arytenoid cartilage. Since the relationship between the arytenoid and the thyroid ala varies in different individuals, it may not be so easy to determine just where to make the window. To overcome this difficulty, the speaker described how he uses a Jackson anterior laryngoscope in a darkened room, by which means the arytenoid can be located through the thyroid cartilage and the exact point for the window outlined. After the window is made the scope is again introduced, the interarytenoideus muscle severed, the arytenoid mobilized by disarticulation from the cricoid cartilage and pulled through the window by means of a suture which is fixed to the perichondrium of the thyroid.

Dr. Mervin C. Myerson described a procedure suggested by Dr. Imperatori which had proven successful in one case. It consists of a simple laryngofissure and the passage of a suture through the posterior end of the vocal cord, carrying it through the cartilage and tying it in the subcutaneous tissues external to the thyroid cartilage. The procedure is simple and avoids the necessity of removing any cartilage. In another case the operation failed because of an ensuing perichondritis.

Dr. Thomas C. Galloway felt that there was no need of removing the arytenoid cartilage when it can so easily be pulled into the window of the thyroid and fixed. He also agreed that the use of the laryngoscope was an advantage in locating the site for the window.

Dr. Francis L. Weille reported having followed a case that had been operated on by Dr. Kelly, and being impressed with the fact that

the patient did not aspirate fluids into the larynx due to the disturbed musculature, and asked Dr. Kelly for an expression on this point.

Dr. Kelly, in closing, stated that as a rule he is opposed to the use of foreign bodies in the larynx, such as the mold mentioned by Dr. Foster, on the ground that the less irritation after either an intra- or extralaryngeal procedure, the better off you are. The suture alone has, in his experience, been sufficient.

Concerning the question as to whether the arytenoid should or should not be removed, Dr. Kelly said he had no quarrel with those who advocate its retention, since he conceded there were different procedures that yielded good results in different hands. He recommends his operation to those who are not satisfied with their previous techniques or those who had no procedure to start with.

Concerning the ability to swallow liquids without aspiration, Dr. Kelly stated that it was astonishing to observe this phenomenon, which to some observers might seem to contradict some of Dr. Negus' theories on the mechanism of deglutition.

Extralaryngeal Surgical Approach for Arytenoidectomy: Bilateral Abductor Paralysis of the Larynx

HENRY B. ORTON, M.D.

NEWARK, N. J.

(This paper appeared in full in the June issue, page 303.)

DISCUSSION

Dr. J. D. Kelly stated that it was important not only to provide the patient with an adequate airway, but also to see to it that he retained some sort of a voice. He prefers to remove the arytenoid, since retention of this organ makes it necessary to separate the cords so far that the vocal function is definitely impaired. Removal of the arytenoid provides adequate breathing space without the necessity of dragging the vocal cord so far laterally as to prevent phonation. Dr. Kelly admitted that Dr. Orton's operation was simple and could be done without making a transverse incision or cutting the pretracheal muscles. However, removal of one third of the posterior part of the thyroid cartilage and sewing the cord out result in a great deal of scar tissue formation, which is likely to pull on the posterior part of the larynx, thus partially blocking that region. Dr. Kelly ascribes the

success of his own operation to preservation of the thyroid cartilage, which Dr. Orton removes in part.

Dr. Lawrence R. Boies reported a successful result in a 37-year-old woman, who was operated upon by Orton's technique. The voice was not quite as good as previously, but the airway was very satisfactory. He was impressed with the simplicity of the procedure.

Dr. Mervin C. Myerson, amplifying his discussion of Dr. Kelly's paper, stated that the position of the vocal cord is not so much dependent on the location of the entire arytenoid cartilage as on the vocal process. Therefore, his suggestion that the vocal process be severed through a laryngofissure, and the posterior part of the cord pulled laterally by a suture passed through the thyroid cartilage, should serve the purpose with the minimum amount of disturbance to function or the removal of any tissue.

Dr. Orton, in closing, admitted that the voice is not as good after these operations as before, but the same is true of almost any operation on the larynx. As for the scar formation mentioned by Dr. Kelly, he had found that the tendency was to contract laterally rather than medially, provided the mucosa was not damaged.

An Unusual Case of Carcinoma of the Larynx

LOUIS H. CLERF, M.D.

PHILADELPHIA, PA.

(This case report appeared in full in the June issue, page 335.)

Fibrosarcoma of the Larynx

J. H. FOSTER, M.D.

HOUSTON, TEXAS

(This case report appeared in full in the December issue, page 764)

Congenital Dermoid of the Pharynx

J. H. FOSTER, M.D.

HOUSTON, TEXAS

(This case report appeared in full in the September issue, page 578.)

Neurofibroma of the Nasopharynx

F. T. Hill, M.D.

Waterville, Me.

(This case report appeared in full in the June issue, page 326.)

DISCUSSION

Dr. J. H. Foster mentioned a similar case he had reported several years ago, and commented on the ease with which the tumor shelled out and the comparative absence of bleeding. A Horner's syndrome was present for a few days after the operation.

Dr. Andrew A. Eggston reported his observations in a case operated on by Dr. Negus about a year ago. The tumor had been located on the left side behind the pillar and was responsible for a paralysis of the recurrent laryngeal nerve. It shelled out easily and, like Dr. Foster's case, was followed by a Horner's syndrome.

Dr. R. S. Moore cited a similar tumor he had removed from the larynx about 15 years ago. There has been no recurrence to date.

Atresia of the Pharynx Following Tonsil and Adenoid Operation

Charles J. Imperatori, M.D.

New York, N. Y.

(This case report appeared in full in the June issue, page 329.)

DISCUSSION

Dr. Thomas E. Carmody mentioned a procedure in which a flap from the cheek or the oral side of the palate is turned under to cover the raw surface after the palate has been released. This procedure, does not cover the raw area on the posterior wall of the pharynx. In a case which he expected to operate on soon, Dr. Carmody planned to use a split graft.

Dr. Gordon Berry stated that he was under the impression that the procedure described by Dr. Imperatori antedated Dr. MacKenty, having been previously described by Dr. Rowe of Rochester.

Dr. Imperatori, in closing, stated that he claimed no originality for the operation, but had been under the impression that it was first described by MacKenty.

Congenital Lymphangiomatous Macroglossia, with Cystic Hygroma of the Neck

D. M. LIERLE, M.D.

IOWA CITY, IOWA

(This case report appeared in full in the September issue, page 574.)

DISCUSSION

Dr. Charles Imperatori recalled a similar case he had reported before this Society in 1940 or 1941, in which the child suffered so much from dyspnea that the only way it could breathe was with its head over the end of a pillow, similar to the Rose position. This case was treated with aspiration and injection with a sclerosing fluid. The patient recovered.

Malignant Recurring Nasal Hemorrhage of Undetermined Etiology

SAMUEL SALINGER, M.D.

CHICAGO, ILL.

(This case report appeared in full in the September issue, page 583.)

Muscles of Phonation and Deglutition as Viewed Through the Operative Wound in Frontal and Ethmoid Sinus

W. L. SIMPSON, M.D.

MEMPHIS, TENN.

(This case report appeared in full in the June issue, page 344.)

Tumor of the Carotid Body

P. P. VIOLÉ, M.D

LOS ANGELES, CALIF.

(This case report appeared in full in the September issue, page 569.)

DISCUSSION

Dr. Walter B. Hoover stated he had recently seen two cases closely resembling that described by Dr. Violé. One case in a man of 30 was unsuccessfully operated on by both external and internal approach. The other case, in a woman of 56, was a tumor presenting in the pharynx behind the palate, which was pushed forward and extended up to the base of the skull, and also presenting at the angle of the jaw. In this case the common carotid artery was compress-

ed for several days prior to operation so that it could be tied if necessary. However, the tumor was removed without this being done.

In the first case the tumor was removed externally at a second operation. It was found to be highly vascular. In both cases tracheotomy and a feeding tube were necessary for a short time.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, November 6, 1944

THE PRESIDENT, DR. ELMER W. HAGENS, IN THE CHAIR

Practice of Otolaryngology In China In Peace and War.

A. M. DUNLAP, M.D.

VISITING CLINICAL PROFESSOR IN OTOLARYNGOLOGY, UNIVERSITY OF CHICAGO

(Abstract)

Dr. Dunlap spoke informally of his experiences as an otolaryngologist in China, covering a period of something over 30 years ending with his repatriation on the Gripsholm in December, 1943. During this time China had been in and out of civil war and, finally, in 1937 had been invaded by the Japanese, and this war is still continuing.

Dr. Dunlap had gone to China in the first instance with the Harvard Medical School of China, an organization sponsored by, but without any organic affiliation with, Harvard University Medical School. Experiences with the establishment of the first eye, ear, nose and throat clinic in St. Luke's Hospital of Shanghai were related, setting forth the various diseases and conditions met with. There were periods in which the hospital devoted a great deal of its service to the treatment of wounded soldiers in and around Shanghai.

In Shanghai between 1911 and 1916, acute hemolytic streptococcus infections and acute mastoid cases were rare. Later such cases became prevalent in Peking as a complication of scarlet fever. A colleague of the speaker working in Chengtu, West China, had said that at that time there were no streptococcus mastoid infections in his area. But this same individual in later years said that, following the coming of the airplane to West China, they were having a fair number of this kind of infection. The airplane had reduced travel time from six weeks to five hours.

In 1918 the speaker moved to Peking where a new clinic was established in connection with the Rockefeller Foundation Hospital, the Peking Medical College, and this clinic was devoted entirely to nose, throat and ear cases. Again it was the first of its kind to be established in China.

During this period an excessive number of acute ear infections were seen. Foreign bodies also were in evidence, the most common perhaps being the date seed, which in the North China variety is extremely sharp on each end and frequently lodges in the upper part of the esophagus.

In the early twenties the first experiments were done on ephedrine. Ephedrine comes from a Chinese herb (Ma Huang) used in connection with the treatment of bronchitis and had been noted for many years as being very effective in certain cases. The first actual administration of ephedrine in the clinic was done by a woman doctor assistant, Dr. Alice Cook, and the first applications were made with five and ten per cent solutions, which concentrations were later found to be unnecessary. After experimentation on dogs, a one per cent solution proved to be adequate.

Cancer of the esophagus was found to be extremely prevalent in North China, possibly due to excessive use of a strong native wine. Carcinoma of the nasopharynx, however, was frequent, not being as prevalent as in the southern part of China.

In 1931 the speaker returned to Shanghai in private practice, and as Clinical Professor of Otolaryngology at St. John's University Medical School. During this period the Japanese war began. Experiences were related from the start of the war in 1937 to the declaration of war against the United States by the Japanese on December 7, 1941. While the Japanese were completely in control of Shanghai, foreign doctors had been left pretty much alone while the business men of Shanghai were being systematically eliminated. Finally, however, pressure from higher-ups compelled local authorities to close the offices of all foreign doctors, and they were interned along with other civilians of Shanghai.

Meeting of Monday, December 4, 1944

THE PRESIDENT, DR. ELMER W. HAGENS, IN THE CHAIR

Massive Pharyngeal Hemorrhages—Case Report

GEORGE H. WOODRUFF, M.D.

(This paper appears in full on page 202)

DISCUSSION

DR. MAURICE F. SNITMAN: Generally, thrombosis of a vessel is secondary to an injury to its wall by infection or trauma. When due to infection there is slowing of the blood stream and a change in the blood, a natural protective response to facilitate sealing of the injured vessel wall. Platelets are first laid down with an admixture of white blood cells, this forming a rather firm tenacious coating. Because of the slower circulation in the veins, they are more frequently thrombosed than are arteries.

It is difficult to determine whether pharyngeal hemorrhage occurs from rupture of the internal carotid artery or from one of the pharyngeal branches of the external carotid. A small sudden hemorrhage from the internal carotid artery is possible, since the rupture may be of very small size. Salinger and Pearlman state that most hemorrhages are from the internal carotid artery, while Kerwin believes that most ruptures occur in the pharyngeal branches of the external maxillary artery. The determination of the vessel involved is extremely difficult during surgery on infected neck tissues.

The Army has prepared a film for the medical personnel on the importance of the so-called anterior tubercle of the sixth cervical vertebra. In practically all dissections one finds that the common carotid passes in the vicinity of or just over the anterior surface of this tubercle, so that when sudden hemorrhage takes place pressure should be applied to this region. It is very easy to feel that artery in the neck because at this point it is very superficial.

The question arises why, after ligation of the common carotid artery, there is no hemorrhage. One would expect bleeding to continue because of the connection of the artery with the circle of Willis and with the two verte'ɔral arteries. It has been shown that after tying the internal carotid artery there is a reduction of almost half

the blood flow in the artery, and one can readily see why this would produce good hemostatis. Carmack stated that fatal hemorrhage from the carotid artery must be more prevalent than one can determine from the literature. It would be natural that some cases are not reported.

Dr. Woodruff is to be complimented on the successful management of his case.

DR. R. W. KERWIN: I became interested in this subject while studying under Professor Wesely at the Neumann Clinic in Vienna. He taught us to section the vessel. In looking over the literature, I found a great deal of material on the subject, in which many cases did not terminate successfully as did Dr. Woodruff's. Horsley, Jr., stressed the fact that besides contracting the artery also retracts, and when the vessel is simply ligated, allowance is not made for this second factor of retraction and buttonholing of the vessel is likely to happen. Against that we have the experience of many men at Cook County Hospital, who have simply ligated vessels without sectioning and have obtained apparently satisfactory results. In doing a few selected cases, I sectioned the vessel between two transfixed ligatures, on the advice of Professor Weseley. There was no immediate or delayed bleeding from the ligated ends of the vessels. Then I did a section of the common carotid artery itself, which took a lot of courage. There was no postoperative bleeding, and fortunately none of the cerebral sequelae which supposedly follow such procedures. As a result of this experience I do not hesitate to section an artery of any size, rather than to trust to simple ligation.

DR. THOMAS C. GALLOWAY: I think I reported one of the earlier cases of erosion of the carotid. In this case an assistant could not at all control hemorrhage by compression against the vertebra. It is a practical point that one should first isolate the common carotid and surround it by a ligature. In profuse and sudden hemorrhage in the pharynx it must be assumed that bleeding is from the internal carotid artery itself, since it has no cervical branches except that the ascending pharyngeal may arise at the bifurcation. One must bear in mind that unusual bleeding may come from the internal jugular vein. It may be active and persistent, and although it can be temporarily controlled by packing, if the vein cannot be ligated in the neck above the bleeding, it may require exposure of the sigmoid sinus through the mastoid, and packing down to the bulb.

DR. GEORGE H. WOODRUFF (closing): I was glad to have Dr. Snitman's comment on thrombosis of the veins. The internal maxil-

lary seems to be the vessel involved most frequently in post-tonsillectomy hemorrhage. However, in deep abscess of the neck, all statistics point to the internal carotid as being the usual source of hemorrhage. I purposely did not touch on bleeding after tonsillectomy, although that subject is important.

In reply to Dr. Galloway's comment: As I understand it, the internal carotid lies in the posterior compartment of the parapharyngeal space. It does not run through the pharyngomaxillary space which is further anterior, in fact this term is synonymous with the so-called anterior compartment of the parapharyngeal space.

Meeting of Monday, January 8, 1945

THE PRESIDENT, DR. ELMER W. HAGENS, IN THE CHAIR

Alterations In the Brain Due To Asphyxiation

WILLIAM F. WINDLE, M.D.

PROFESSOR OF NEUROLOGY, NORTHWESTERN UNIVERSITY MEDICAL SCHOOL

(By Invitation)

(Abstract)

Guinea pigs were asphyxiated and resuscitated at birth. The brains of these and of normal litter mate controls were sectioned serially and stained by a method permitting accurate histologic comparison.

Hemorrhages occurred in the brains of all three hours to five days after resuscitation, but none were killed by asphyxia. Edema appeared between eight hours and four days after resuscitation. Hemorrhage and edema apparently were not the primary causes of neuron damage. Nerve cell changes, including peripheral vacuolization and chromatolysis, occurred in all specimens between 1½ hours and 21 days. Marked cytopathology was often circumscribed. The cerebellum and corpus striatum were not severely affected, but the thalamus, cerebral cortex, tegmentum and spinal cord were often badly damaged. A transient microglia proliferation took place at two to 21 days. Generalized or regional atrophy followed neuron

destruction in two thirds of the severely asphyxiated and half of the mildly asphyxiated animals.

It was concluded that experimental asphyxiation followed by successful resuscitation of guinea pigs at birth produced neuropathologic changes of variable degrees of severity in all specimens studied one and one-half hours to three weeks after birth, and produced detectable permanent structural changes in more than half of those allowed to live for more than three weeks.

DISCUSSION

DR. ANDREW C. IVY: The work Dr. Windle has been doing is of obvious interest to physiologists and I believe also to anyone called upon to relieve asphyxia or tracheal obstruction, or who has had an unfortunate anesthetic accident. In our work we have frequently had occasion to operate in the thorax, and in one out of 15 dogs, on sectioning the vagi and the thoracic sympathetic trunks, the respiration and heart have stopped. They do not recover unless something is done. If something is done, you can resuscitate every one of these animals. The best procedure in these (I can speak with experience because I have had 20 such cases) is to inject 0.5 mg. of epinephrine and 1.0 mg. of atropine into the ventricle and apply massage to the heart. When the blood pressure is brought to normal in five minutes, respiration will return along with the blood pressure; such a dog will regain consciousness. But if the blood pressure is not brought back to normal in 10 minutes the dog will never regain consciousness, although one can frequently resuscitate the respiratory centers and vasomotor centers in the dog after 10 minutes. This illustrates how sensitive the cerebrum is to anoxia in the dog. The medullary centers are much more resistant than the cerebrum.

The hemorrhage seen in the brain substance in asphyxial death has interested me from the viewpoint of the explanation. I would like to know how Dr. Windle explains the hemorrhages he has observed. In asphyxia the blood pressure rises in the initial stage; this rise along with asphyxial convulsions may cause a mechanical rupture of the blood vessels; this is the only explanation that occurs to me. Associated with obstructive asphyxia in the dog and frequently in human beings, we have observed beneath the pleura, petechial hemorrhages. We have explained this as being caused by mechanical pressure leading to rupture of the capillaries. So far as I know this is the only explanation applicable to the lung. If Dr. Windle's guinea pigs did not show asphyxial struggles, we would have to search for another explanation.

Of course when there is a hemorrhage and actual destruction of brain cells in the primary motor and primary sensory areas, we can understand why such an animal or human being is unable to learn, but on the basis of recent studies of the function of the frontal area of the cortex, one should be able to get along when deprived of a large number of brain cells located in association areas. I wonder if Dr. Windle ascribes the disturbance of learning in the guinea pig to the lesion of the primary sensory and motor areas of the cortex, or to a disturbance of the association areas of the cortex.

We find that the young animal is much more tolerant to asphyxia and anoxia than the adult. On the basis of our observations that is true. But does that go far enough? We attempt first to resuscitate the circulation and respiration in order to obtain survival, and then we look for a return of consciousness, but as a general rule we do not look further. It may be if we took adults and examined their ability to perform and to learn and then subjected them to asphyxia, we might find that the adult as well as the young animal is sensitive to asphyxia. It may be that asphyxia in the adult will interfere with learning. At present we cannot conclude that the cerebrum of a young animal is more resistant to asphyxia than the cerebrum of the adult, because we have not measured the adult. Is there any evidence to show that the vital centers in the medulla of the newborn guinea pig will withstand complete ischemia longer than the medullary centers of the adult guinea pig? In the adult dog the figure is 20, 30, 35 minutes of ischemia. I have not observed the medullary centers of puppies withstand asphyxia longer than 30 minutes.

In the adult man it is interesting to know that when the oxygen saturation of the hemoglobin of the blood is reduced to 80 or 85 per cent, impairment of vital function occurs; if the saturation of the blood is reduced to 66 per cent, the majority of individuals will lose consciousness. What does that mean? It means that 5 of the 15 grams of hemoglobin in the blood are reduced hemoglobin; this is the condition normally present in the venous circulation; or the hemoglobin returning to the right heart is approximately 66 per cent saturated. That is, an adult man may lose consciousness when the arteries contain that amount of reduced hemoglobin which is normally present in venous blood. This is a good way to remember the amount of oxygen in the arterial blood required to maintain consciousness.

Dr. Windle's observations emphasize the importance of relieving a tracheal obstruction as early as possible, not only to save life but to

prevent irreversible damage to cortical nerve cells. In the case of anesthetic accidents, speed is indicated not only to save the life of the patient, but to preserve the normal mental activity of the patient.

DR. THOMAS C. GALLOWAY: The clinical implications of this work are great. We have considered anoxia rather abstractly as it occurs in many clinical conditions, and we have wondered how much of the picture was due to anoxia. I know no work comparable to this as a clear-cut demonstration of what anoxia does to the brain tissues. The importance of anoxia in bulbar poliomyelitis, in tracheobronchitis, in anesthesia, in carbon monoxide poisoning, in flame and gas injuries and many other conditions is, I am sure, frequently not appreciated.

Recently at a symposium on poliomyelitis, one of the pathologists gave some beautiful demonstrations of his idea of histopathology of the disease. His photomicrographs were much like those Dr. Windle showed, and I think much of his picture might be due to anoxia. In clinical descriptions of this disease many symptoms ascribed to virus infection such as depression, disorientation, restlessness and even coma may change very quickly when the airway is cleared and seem almost certainly due to anoxia.

Anoxia can so quickly become irreversible that what we do must be done quickly. Respiratory obstruction must be relieved before permanent damage has been done, and the margin of safety may be slight. If the brain suffers so quickly from anoxia, other organs must also be damaged, especially the heart and the respiratory muscles.

DR. PAUL HOLINGER: Certainly many clinical phenomena have been explained by this most excellent work that Dr. Windle has done and has shown us today. I should like to ask him if he has had any experience in producing similar anoxemias to those shown by overdosage of barbiturates. These sedatives are often used in excessive amounts and severe respiratory obstruction results. It would appear to me that their use, or abuse, in obstetrics and in pediatrics could lead to conditions such as were demonstrated here.

DR. ELMER W. HAGENS: Many times it is important to decide whether the difficulty is due to a central or a peripheral lesion, and whether it is possible to separate them. We are also interested in whether to use oxygen or carbon dioxide, or a combination. I would like to ask about artificial respiration. It seems that some

patients recover more rapidly by a combination of oxygen with active movements stimulating respiration.

DR. WILLIAM F. WINDLE: I have no really good answer to the question, why hemorrhages occur. In our observations it appeared that hemorrhage did not occur until after resuscitation was effected. A few animals which were not resuscitated but asphyxiated to death were autopsied; the brain was studied histologically and we found no hemorrhages. In some of those that lived but a short time we found no hemorrhages. In all those that were resuscitated and lived three hours or more we did find hemorrhages. So it seemed that the hemorrhages came about after resuscitation. We simply guessed that after resuscitation, with a rise in blood pressure and perhaps accompanying weakening of the capillary walls—though how, I do not know—we got a leakage of blood. We do not think the hemorrhage occurred because of the struggle during asphyxiation, as the struggle was not marked.

DR. ANDREW C. IVY: It has been shown that asphyxia increases the permeability of the capillaries to protein but, according to my information, it has not been shown that asphyxia permits the red cells to pass out by diapedesis. It is possible that asphyxia might alter the capillaries of the brain and lungs to such an extent that hemorrhage *per diapedesis* results.

We are in process of investigating various methods which have been recommended for resuscitation. So far as my interpretation of the literature is concerned, it is rational to use 5 to 10 per cent carboxygen, i.e., carbon dioxide with 90 to 95 per cent oxygen, in the early treatment of carbon monoxide posioning, because the evidence suggests that when carboxygen is used the carbon monoxide is cleared from the blood more rapidly than when 100 per cent oxygen is used. The only reason in my opinion for the use of carbon dioxide is to stimulate the respiratory centers and to treat the effects of acarbia when due to hyperventilation; but you cannot always tell when the center is depressed in such a way that it is advisable to stimulate it with oxygen.

I cannot with conviction based on evidence tell you in which conditions other than carbon monoxide poisoning it will be beneficial to administer carboxygen. There is one point that should be kept in mind. If you should breathe a mixture of 5 per cent carbon dioxide and 95 per cent oxygen, you would be tired out in 30 minutes as a result of extra respiratory effort, and you could perform but little work while breathing the mixture. One can breathe 3 per cent

carbon dioxide and 97 per cent oxygen for an hour or more without becoming tired. So, if you have a patient already weak and exhausted and give 5 per cent carbon dioxide and oxygen for longer than a few minutes, you add to the exhaustion. I have taught for more than five years that if carboxygen is used to clear the lungs after an anesthetic it should not be used longer than five or ten minutes. If you are skeptical of the truth of this point, I suggest you breathe five or seven per cent carboxygen; three per cent carboxygen for longer periods and five or seven per cent for short periods may be beneficial in certain states of "shock" but in my opinion there is not enough evidence in the literature to be dogmatic about it. We need well-controlled evidence from the laboratory and clinic. There is a paucity of such evidence at present.

DR. WILLIAM F. WINDLE (closing): A number of observations have been reported with regard to the survival time. It has been shown in rabbits and rats, and we have shown it in the cat, that the survival time—the time during which an animal will live while in an atmosphere of nitrogen or drowning under water—is greater for the newborn or the fetus than the adult. An adult rat in an atmosphere of nitrogen will die in two or three minutes, whereas a newborn rat will live for nearly an hour. In the case of the newborn cat, the survival time appears to be about 30 minutes. For the guinea pig it is less; 20 or 22 minutes is the maximum.

Dr. Galloway mentioned damage of other organs; we did find hemorrhage in other organs, notably the lung and some of the abdominal organs, even on the wall of the stomach and the liver.

Dr. Hagens asked whether to use oxygen or carbon dioxide. In the case of the newborn, I think you are justified in using any means at hand; the quickest is the best. Do not wait for oxygen or a drug. The main thing is to get air in, by mouth-to-mouth breathing if necessary, in the case of an infant in dire distress.

At one time we felt there was a good deal of justification for the use of carbon dioxide with oxygen, and we did some experiments in which the pregnant cat was given nembutal in large amounts, and the fetuses resuscitated afterward. We had little success with carbon dioxide and oxygen, but oxygen alone gave better results. The present experiments were done without anesthesia, except local. In some experiments we asphyxiated two fetuses and resuscitated one with carbon dioxide and oxygen mixtures and one with oxygen alone. It did not seem to make much difference.

There were parts of the brain in which we found no hemorrhages in any animal; there were none in the cerebrum or corpus striatum. The incidence of hemorrhage was extremely variable. The thalamus was the most frequent site.

The Therapeutic Value of Lipiodol In Sinus Infection

HANS BRUNNER, M.D.

(Abstract)

Lipiodol usually does not exert any influence whatsoever upon the nasal mucosa; nor does it cause a permanent paralysis of ciliary action.

If introduced into the maxillary sinus, the oil goes to the bottom if a sufficient amount is used; it must be heavier than the exudate in the sinus.

Conclusions concerning the thickness of the mucosa should not be drawn from films taken immediately after the injection of oil, unless a proper amount of oil was introduced.

Filling by displacement usually does not permit the introduction of an adequate amount of oil into the maxillary sinus.

Assuming that iodized oil offers any therapeutic effect, this consists only in protecting the membrane against the action of the exudate and in breaking the vicious circle of exudate and membrane.

In chronic infections of the maxillary sinus there are two types of emptying; in the first, the emptying is definitely delayed, but the emptying time approaches normal if the chronic infection begins to subside. In the second type the emptying time is normal or hastened.

In the first type, emptying is chiefly through ciliary action; in the second, by exudation pressure.

A thorough tampon effect by the oil can be achieved only in the first type; but it seems that lipiodol hastens cure in the second type also.

DISCUSSION

DR. T. J. WACHOWSKI: So far as radiologists are concerned we have to depend solely on objective findings, and these are subject to differences of opinion as to interpretation. They do not take into con-

sideration subjective findings or symptoms, and many objective findings such as drainage or pus, color the interpretation of the clinician. We do not have the benefit of that. It might be of interest to say that when we did some work on conscientious objectors we found a high percentage of what we would call evidence of diseased sinuses. Most showed no symptoms or complained only of postnasal drip, which we would expect in this climate. What we might say represents active pathology, and what you might say in reading the same film, might vary considerably.

One thing Dr. Brunner showed is the oil floating in the exudate. We see the same thing in the oviduct; the oil spills out of the tube and you might think it is patent. You will see that in 24 hours the oil is still in the same position. Certainly it is presumptuous to say the oil should reach the bottom in a few minutes. Most oil will go down through pus and I think Dr. Van Alyea has some interesting things to show in that respect.

The question of a defect between the rim of the antrum and the oil comes up. I wonder if that space is not due to thickened membrane. I realize that a membrane can get very thick, and certainly in acute infections we see this due to edema. I wonder whether part of the decrease of that thickening is not due to the fact that Dr. Brunner has established drainage and whether that has not had a therapeutic effect. I know how rapidly such regression can occur.

I should like to report two patients:

The first patient had an oil filling showing a marked hyperplasia of the membrane of the antrum. No further oil was instilled but drainage was maintained for three months. A repeat oil filling at that time showed an approximately normal lining membrane. Such results are not unusual and can be attributed almost entirely to the drainage which is established.

The second patient had an oil filling showing a marked hyperplasia of the lining membrane of the antrum. Following this procedure oil drained for a few days, but then the drainage stopped. He suffered severe pain for two or three days which necessitated a narcotic. Following that, the symptoms regressed and he has been followed for nine months by x-ray films which reveal a persistence of the thick membrane and a large residuum of oil in the antrum. From the standpoint of the patient, he is well; therefore he has had nothing done to the sinus. Whether we have a patient who is very phlegmatic and refuses to admit he has pathology, or whether you can have the

appearance of marked infection in the sinus without symptoms, I cannot say. It would seem that the oil alone, without the benefit of drainage, has not effected an orthodox cure. It is obvious that the sinus is not emptying and one would expect severe symptoms, but he disclaims them. It is possible that the lipiodol has something to do with this unusual situation. Dr. Van Alyea is familiar with the case and I should like him to explain the confusing picture.

DR. O. E. VAN ALYEA: Dr. Brunner has presented some very interesting angles to the subject of lipiodol in sinus therapy. Dr. Goodyear, back in the middle twenties, noted improvement in the sinuses he was studying while using oil as a diagnostic aid. Since then he has kept the subject alive with many articles, his most recent paper being presented at the 1940 meeting of the American Medical Association.

In discussion of that paper, Dr. Proetz brought out the idea that the oil may serve as a protective membrane for the sinus mucosa. He noted that when oil was poured into a test tube containing a watery solution, it settled to the bottom. During the past few days I have repeated this experiment, using samples of exudate obtained from infected sinuses. Various types of sinus exudate were collected and in all cases the oil was soon seen at the bottom of the tube. In the watery type of secretion, that known as the "pea soup" variety, the oil passed through immediately. In some of the thick, heavy exudates, the penetration time was approximately 30 minutes.

The value of oil as a therapeutic agent is the subject under discussion. Dr. Brunner has followed up the suggestion of Dr. Proetz that the oil may act as a protective tampon. I quite agree with this. The value of such protection is more clearly appreciated when the nature of the inflammatory exudate is understood. The polymorphonuclear leukocytes present in large numbers contain powerful proteolytic enzymes, the purpose of which is to dissolve phagocyted bacteria. In a stagnant exudate, such as may be commonly found in the infected maxillary or sphenoid cavities, the pus cells disintegrate. Their content of enzyme is thus released and acts as an irritant to any tissue with which it may come in contact. In poorly draining cavities, the toxic effect of this enzyme may well account for a prolongation of infection. If we can assume that this oil displaces that enzyme, removing it from contact with the mucosa, we have in truth a tampon, one which may well contribute to the elimination of a persistent infection. It is important, however, that the exudate have a chance to get out of the sinus. To depend entirely

upon the instillation of a small quantity of oil, as advocated by Goodyear, is asking a great deal of a single procedure. With sinus infection there is inadequate drainage; proper management, therefore, includes all necessary measures to correct drainage defects. This may require certain minor surgical procedures and careful analysis of the patient's general condition, including an allergy survey. An occasional sinus lavage is helpful not only for the purpose of removal of accumulated secretions but to throw light on the progress of the case.

Over-irrigation is harmful, as stressed by Dr. Brunner. It is quite true that infection may be kept in an active state by daily irrigations. I have found that sinus infections tend to clear up more rapidly when an interval of one week is allowed between treatments, whether this consists of some minor surgical corrective measure or merely sinus lavage.

Dr. Samuel Salinger: Dr. Brunner has a way of making a subject appear extremely simple and at the same time very complicated. When we speak of chronic sinusitis, what kind of sinusitis are we talking about? I recognize three types: a chronic sinus in which the mucous membrane is thickened but in which I rarely find any secretion; another type in which there is a very profuse discharge; and a third type characterized by the formation of polypi and thick mucoid or mucopurulent secretion. I cannot conceive of a therapeutic procedure that is equally applicable to all three. My own experience has been (and I use lipiodol a good deal for diagnosis) that in cases in which the lipiodol has failed to be eliminated from the sinus within a couple of weeks, that sinus will not get well without surgery. I have gone in three to six months later and found beads of that lipiodol encysted and inspissated, and remaining as a foreign body. So it is difficult for me to accept the tampon theory. Despite the fact that patients have said they felt better and I have noted a diminution of secretion, I cannot say that I have found them to be cured if they had an irreducible infection in the mucous membrane. I think if we are evaluating therapeutic procedures we should be more specific about the type of pathology we are discussing.

Dr. Andrew C. Ivy: In regard to the patient in whom the lipiodol remained in the sinus for a prolonged period, did you introduce a second quantity to ascertain how long it would remain? If the patient's sinuses were normal, as you assumed, a second injection of lipiodol should have been discharged in a few days.

DR. WALTER H. THEOBALD: Concerning the functional behavior of the maxillary sinus, there is one point Dr. Brunner did not mention. He stated that the lipiodol does not sink to the bottom in some cases but floats on top of the exudate. These test tubes show it going to the bottom, however. The simplest procedure is to inject lipiodol after irrigating the sinus, thereby eliminating all the exudate. In 1933, we made some investigations with radiopaque oil, injecting it into the antrum while keeping the head in a lateral position and taking films, lateral and anteroposterior, in the same position. This kept the oil in situ and revealed complete filling of the antral cavity.

In 1936 King presented a paper on ciliary function within the antrum on postoperative cases. In like manner, I studied the ciliary activity of a known normal maxillary sinus, and noted the rapidity with which a radiopaque substance is removed from the antrum. For this purpose we used barium as a radiopaque substance, with the idea that it might provide a more nearly normal response from the cilia. Therefore, 1 cc. of a heavy mixture of barium sulphate in water was injected into the antrum through the natural opening, without the use of cocaine or other medication. Study of the films revealed a prompt migration in the direction of the natural opening within 25 minutes. Further exposures on succeeding hours and days showed that it required eight days to empty the antrum completely.

In another case in which a radical operation had been performed three and one-half years previously, this functional test revealed a small diminution of barium after two and a half days. Nine days later x-ray films showed only a slight decrease in the amount of barium. From this observation it would appear that the antrum mucosa ciliary function was absent. Despite this fact there had been no recurrence of infection in this case.

DR. HANS BRUNNER (closing): In answer to Dr. Ivy, in the case to which he referred, no additional lipiodol was introduced during the nine weeks of observation.

I believe Dr. Wachowski misunderstood me. I considered the large space between the floor of the sinus and the lipiodol as swollen mucosa and not as exudate. But I emphasized that this swollen mucosa on the floor of the sinus, together with the exudate, is capable of pushing the lipiodol toward the natural ostium and thus expelling it from the sinus. Due to this mechanism, the emptying time in acute sinus infections is normal or shortened, while in chronic sinus infections it is delayed, when achieved by ciliary action. This was the principal finding in my report.

I do not know that there are different clinical types of chronic sinus infections, if the specific infections are put aside. The sinus mucosa may react in different manner to chronic infection, but these are histologic and not clinical varieties. Certainly I do not know the rationale of introducing lipiodol into a sinus for therapeutic purposes if the sinus contains only hyperplastic mucosa but no exudate.

SIR JAMES DUNDAS-GRANT, K.B.E., M.D., F.R.C.S.

1854-1944

Sir James Dundas-Grant died in London on November 13th. For many years his name was prominent in the educational field of otolaryngology. He was well known to many Americans of the last generation who studied abroad, and the American Laryngological Association made him a Corresponding Fellow in 1901. At that time he had been practicing the specialty for 15 years.

James Dundas-Grant was born in Edinburgh in 1854 and was educated there, first at the Academy and later at the University where he took his Master's degree in 1873. He studied medicine at St. Bartholomew's in London and became a Fellow of the Royal College of Surgeons of Edinburgh in 1884 and of the English College in 1890. He was connected with various hospitals; the Cancer Hospital, the West End Hospital for Diseases of the Nervous System, the Freemasons' Hospital at Hammersmith, the Sussex Throat and Ear Hospital at Brighton, and the Brompton Hospital for Consumption. He identified himself chiefly with the work of the Central London Throat and Ear Hospital, to which he was appointed in 1879 and in which he remained active until his later years.

His contributions to the literature were many and various. He introduced many new instruments and refinements of old ones and was a prominent figure in the deliberations of the special societies.

He was active in numerous hospitals during the First World War and was the medical officer of a brigade of the London Division of the National Reserve. Between 1917 and 1920 he was identified with the otological activities of the Ministry of Pensions. He was knighted in 1920.

Besides his membership in the American Association he was also associated with several laryngological bodies on the continent. He was at various times President of the Section of Laryngology and Otology of the British Medical Association, and of the Otological and the Laryngological Sections of the Royal Society of Medicine, of the British Laryngological Association, and of the Hunterian Society.

IRA FRANK, M.D.

1877-1945

Dr. Ira Frank, a familiar figure in Chicago and national otolaryngological circles for over forty years, died January 20, 1945. He is survived by his wife, Mrs. Ira Frank, a daughter, Mrs. Lester R. Wellman, and a son, Ira, Jr., of Highland Park, Illinois.

Dr. Frank was born in Buffalo, New York, in 1877 and was graduated in 1899 from the College of Physicians and Surgeons, Chicago, now the College of Medicine, University of Illinois. After completing an internship at Michael Reese Hospital, he spent two years in Vienna developing his specialty with the masters of those days and returned to Chicago, where he engaged in active practice until his retirement in the spring of 1943.

Dr. Frank was certified by the American Board of Otolaryngology and was a member of the following societies: Chicago Laryngological and Otological Society of which he was President in 1936-1937; Chicago Medical Society, Institute of Medicine of Chicago; Chicago Society of Internal Medicine, American Academy of Ophthalmology and Otolaryngology, Society of Medical History of Chicago, American Laryngological, Rhinological and Otological Society, and Illinois State Medical Society.

He was ever alert to the newer developments in medicine, was a regular attendant at scientific meetings and his contributions to the literature covered a wide range of interesting and instructive subjects.

After his retirement Dr. Frank was allowed time to indulge freely in the activities which interested him. These included fishing, fly-casting, photography and horticulture. He had a keen interest in music and his house was filled with a fine collection of paintings depicting his artistic attainments. Another interest which occupied his time in later life was that of birds for which he had established a refuge on the grounds surrounding his home in Highland Park.

It might be said of Ira Frank that he had the attributes which approach the ideal in a busy practitioner of clinical otolaryngology. Certain of these attributes were well described in a tribute paid Dr. Frank by an understanding and lifelong friend, J. Christian Bay, Librarian of the Crerar Library, who wrote: "He had grace of manner. He moved lightly and spoke gently and would draw out a his-

tory by small and casual talk. His instinct for the mentality of a most varied clientele was instinctive. He was punctilious in not wasting the time of his patient any more than his own—and yet, while swift and sure of every movement he never seemed under a strain.

"As the war progressed his ever-increasing duties, both those required and those self-imposed, prevented him from gaining leisure enough to concentrate upon his beloved subjects of art in its relation to anatomy and pathology or medical ideas and theories reflected in the work of artists. Demands upon his time increased, yet he remained on deck as long as he was able to keep on his feet, and he will remain on deck in the memory of the thousands for whom he made life easier and whom he would befriend with his great skill as a physician and his cheerful grace as a man."

O. E. VAN ALYEA.

PUBLICATIONS OF DR. IRA FRANK

1. Pollen Extracts and Vaccines in Hay Fever, with Solomon Strouse, J. A. M. A., March, 1916.

2. Pollen Extracts and Bacterial Vaccines in Hay Fever, with Solomon Strouse, J. A. M. A., May, 1919.

3. Nondiabetic Acidosis with Special Reference to Postoperative Acidosis in Children, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, December, 1916.

4. A Report of two Severe Cases of Ulceromembranous Angina, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, September, 1916.

5. Dentigerous Cysts, with Report of a Case, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, December, 1917.

6. On Lung Abscess as a Sequel to Tonsillectomy, Laryngoscope, June, 1917.

7. Gasparo Tagliacozzi and his Contribution to Rhinoplasty, with Mortimer Frank, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGYY, June, 1918.

8. Primary Principles of the Technique of Plastic Surgery of the Face, Laryngoscope, August, 1918.

9. An Invisible Scar Method in Cosmetic Nasal Surgery, with Jerome F. Strauss, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, September, 1921.

10. A Study of the Action of Calcium in Experimental Cocain Poisoning, with Jerome F. Strauss and William A. Smiley, J. A. M. A., October, 1921.

11. Recent Nasal Fractures, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, September, 1923.

12. Retropharyngeal Abscess, J. A. M. A., June, 1921.

13. Pulmonary Abscess following Tonsillectomy, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, June, 1932.

14. The Nasal Mucous Membrane as an Indicator of Faulty Body Chemistry, The Pale Septum, Laryngoscope, January, 1933.

15. The Influence of Sir Felix Semon on the Development of Laryngology, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, September, 1933.

16. The Deafness of Beethoven, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, June, 1935.

17. Spontaneous (Non-Traumatic) Atlanto-Axial Subluxation, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, June, 1936.

18. Hemorrhages Complicating Deep Pharyngeal Infection, with Arthur F. Abt, J. Ped., July, 1936.

19. Granulocytic Angina. Report of Three Cases with Two Fatalities, Arch. Otolaryng., March, 1936.

20. Sheath of the Internal Carotid Artery: A Route for Infections from Primary Lesions, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, December, 1937.

21. Papilloma of the Tonsil—With Report of Three Cases, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, September, 1938.

22. Experimental Endocarditis in Dogs, Arch. Path., March, 1939.

23. Role of the Tonsils in Experimental Endocarditis, Arch. Otolaryng., March, 1939.

24. Carcinosarcoma of the Larynx, with Maurice Lev, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 43:113, 1940.

25. Pyogenic Granuloma of the Nasal Fossa, with Margery Blahd, Arch. Otolaryng., 31:919, 1940.

26. Transitional Cell Carcinoma of the Upper Respiratory Tract, with Maurice Lev and Margery Blahd, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 50:393, 1941.

27. Experimental Hypersensitivity of Mucous Membranes of the Upper Respiratory Tract, with Margery Blahd, Arch. Otolaryng. 35:918, 1942.

28. The Reaction of the Normal and the Allergic Sinus to Virulent Pneumococci, with Margery Blahd and Katharine M. Howell, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 52:81, 1943.

Books Received

Eye, Ear, Nose and Throat Manual For Nurses.

By Roy H. Parkinson, M.D., F.A.C.S., Head Oculist and Aurist to St. Joseph's Hospital, San Francisco, Calif. Fifth edition. Pp. 1-247, with 82 illustrations, including 2 in color. St. Louis, The C. V. Mosby Company, 1944. (Price $2.25)

An elementary treatise on the anatomy of the eye, ear, nose and throat and the common maladies which affect these areas. A few short paragraphs are devoted to the physiology of the respective parts and certain conventional therapeutic measures are suggested.

In the chapter on operating-room technic, operative procedures are described which should be useful to the beginner when modified by individual hospital routines. Several good illustrations are helpful in conveying the author's ideas to the reader. He has made a sincere effort to encompass a subject in a simple manner and has succeeded in avoiding the usual pitfalls and ambiguities of technical descriptions. The subject matter is brought into good focus by the questions at the end of each chapter, which serve not only as a review but also as an incitant to more extensive reading on the part of the student.

McMAHON.

Speech Reading—Jena Method, A Textbook with Lesson Plans In Full Development For Hard-of-Hearing Adults and Discussion of Adaptations For Deaf and Hard-of-Hearing Children.

By Anna M. Bunger, Instructor of Speech Reading, Department of Special Education, Michigan State Normal College, Ypsilanti, Michigan. Revised Edition. The Interstate Special Education Series, Wendell Johnson, Editor. Pp. 136. Danville, Illinois, The Interstate Publishers, 1944. ($2.50)

The present edition of this textbook on speech reading (lip reading) is an elaboration of the first edition published in mimeographed form in 1932. The original edition has been improved by the suggestions received from the many teachers and students who have used the Jena Method as presented at that time by the author. It deals with the basic principles and methods of teaching speech reading. Special attention is devoted to details as well as to well-planned drill exercises for practicing these details. It is recommended by the editor "not only to specialized teachers of speech reading, or lip reading, but also to speech correctionists, teachers of speech, psychologists and educators."

McMAHON.

Notices

UNIVERSITY OF CINCINNATI

Under the auspices of the Departments of Otolaryngology and Anatomy, the College of Medicine of the University of Cincinnati offers its Annual Course in Otorhinolaryngology with work on the cadaver, April 30 to May 5, 1945. Address: Dean, College of Medicine, Eden and Bethesda Avenues, Cincinnati 19, Ohio.

WASHINGTON UNIVERSITY

Washington University School of Medicine announces an eight months' course in otolaryngology beginning September 17, 1945 open to graduates of approved medical schools who have completed a year of general internship. For further information address The Registrar, Washington University School of Medicine, St. Louis 10, Missouri.

AMERICAN BOARD OF OTOLARYNGOLOGY

The American Board of Otolaryngology will conduct examinations on the following dates:

June 5-8, 1945, Hotel Waldorf-Astoria, New York, New York.

October 3-6, 1945, Palmer House, Chicago, Illinois.

All communications should be addressed to the Secretary, Dr. Dean M. Lierle, University Hospital, Iowa City, Iowa.

ANNALS PUBLISHING COMPANY

The Annals Publishing Company wishes to purchase copies of the September 1944 issue now out of print, and will pay $1.00 per copy for any received at the business office, 7200 Wydown Blvd., St. Louis 5, Mo., before June 1.

OFFICERS

OF THE

NATIONAL OTOLARYNGOLOGICAL SOCIETIES

American Academy of Ophthalmology and Otolaryngology

President: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
President-Elect: Dr. Alan C. Woods, Johns Hopkins Hospital, Baltimore, Md.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Palmer House, Chicago, October 7-11, 1945.

American Broncho-Esophagological Association

President: Dr. Carlos E. Pitkin, Carnegie Medical Bldg., Cleveland, Ohio
Secretary: Dr. Paul Holinger, 700 N. Michigan Ave., Chicago 11, Ill.
Meeting: Cancelled

American Laryngological Association

President: Dr. Harold I. Lillie, Mayo Clinic, Rochester, Minn.
Secretary: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis 8, Mo.
Meeting: Cancelled

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Albert C. Furstenberg, University Hospital, Ann Arbor, Mich.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Meeting: Cancelled

Sections:

Eastern—Chairman: Dr. Oram R. Kline, 414 Cooper St., Camden, N. J.
Southern—Chairman: Dr. Verling K. Hart, 106 W. 7th St., Charlotte, N. C.
Middle—Chairman: Dr. Carl H. McCaskey, 608 Guaranty Bldg., Indianapolis, Ind.
Western—Chairman: Dr. Aubrey G. Rawlins, 384 Post St., San Francisco, Cal.

American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia 2, Pa.
Secretary: Dr. Fletcher D. Woodward, 104 E. Market St., Charlottesville, Va.
Meeting: Cancelled

American Otological Society

President: Dr. Gordon Berry, 36 Pleasant St., Worcester, Mass.
Secretary: Dr. Isidore Friesner, 101 E. 73rd St., New York, N. Y.
Meeting: Cancelled